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PAROXYSMAL NOCTURNAL HAEMOGLOBINURIA IN A SOUTH AFRICAN BANTU PATIENT

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Paroxysmal nocturnal haemoglobinuria (PNH) is a rare disease, and we are not aware of any description of proved cases in the Bantu. The clinical manifestations are not always typical, and the disease should be included in the differential diagnosis of patients with haemoglobinuria or apparent haematuria. In this paper we record the findings in a Bantu patient in whom the diagnostic criteria of PNH were fulfilled.

These criteria are: increased rate of haemolysis (as shown essentially by the demonstration of diminished red-cell life-span), haemosiderinuria, and haemolysis of red cells in acidified serum. Certain other features contribute to the diagnosis. The red cells haemolyse when incubated in serum containing high-titre cold antibodies, and haemolysis in acidified serum is enhanced by the addition of thrombin. Clotted blood from patients with PNH haemolyses rapidly when incubated at 37°C. PNH red cells are often deficient in the enzyme acetylcholinesterase. All these features have been demonstrated in the present case.

METHODS

The standard haematologic methods used in this investigation were those described by Dacie.[1]

Ham's acid-serum test: $\frac{1}{10}$ volume of a 50% suspension of washed red cells was added to normal compatible serum acidified by the addition of $\frac{1}{10}$ volume of 0·2N-HCl. The mixture was incubated at 37°C. for 2 hours, and the amount of haemolysis in the supernatant read quantitatively. Controls included normal red cells, unacidified serum, and inactivated serum.

The Crosby test was performed as originally described,[2] and the amount of haemolysis read quantitatively. Controls were set up with normal red cells.

Haemolysis by high-titre cold antibodies: 0·25 ml. volumes of a 10% suspension of test and control red cells were added to 0·25 ml. of a 1 in 25 dilution in normal serum of the serum containing high-titre cold antibodies. The mixture was left at room temperature for 2 hours, and the amount of haemolysis read quantitatively.

Heat-resistance test: Blood from the patient and controls was allowed to clot at 37°C. for 2 hours, and the amount of haemolysis in the serum was measured quantitatively.

Red-cell acetylcholinesterase was measured by the method of Michel.[3]

Neutrophil alkaline-phosphatase activity: Fresh blood smears were stained by the Gomori technique.

Red-cell survival was measured with radioactive chromium, as described by Mollison and Veall.[4] The half chromium time was estimated, and the mean cell life calculated after correction for elution ($T\frac{1}{2}$ elution 64 days).

CASE REPORT

A Bantu (Swazi) female, aged 38, was admitted to Baragwanath Hospital on 29 June 1960, complaining of the intermittent passage of blood in the urine over the previous year. She stated that the colour of the urine varied from pale red to almost black. She had recently had backache, lower abdominal pain with burning and frequency of micturition, persistent frontal headache, mild exertional dyspnoea and fatigue. The family history was non-contributory. Physical examination revealed no abnormality other than pallor of the mucous membranes and the palms of the hands.

Laboratory Findings

Blood count. On admission the haemoglobin value was 7·5 G. per 100 ml., packed-cell volume 25% and mean corpuscular haemoglobin concentration 30%, with reticulocytes 16%. Leucocytes were 2,300 per c.mm. with neutrophils 23%, monocytes 8%, lymphocytes 68%, eosinophils 0% and basophils 1%. On the stained films the red cells showed moderate anisopoikilocytosis, and the platelets were not obviously reduced in number.

Urinalysis. Microscopic examination of the centrifuged deposits from numerous specimens of urine did not show more than 5 red cells per high power field. Protein was present ++, but bilirubin was not detected, and urobilin varied from a normal amount to moderate excess. Methaemoglobin was present in almost all specimens examined. Staining of the centrifuged urinary deposit with Perl's reagent showed the presence of numerous iron granules. The amount of iron excreted in a 24-hour specimen of urine at a time when haemoglobinuria was not present, was 3·6 mg. (normal 30—65 μg.). Cystoscopy on 6 July showed puffs of reddish-brown pigment emerging from the ureteric orifices. Retrograde pyelography revealed normal calyces and pelves, and the bladder was normal.

Blood chemistry. Blood urea was 30 mg. per 100 ml. Total serum bilirubin varied from less than 1·0 mg. to 1·3 mg. per 100 ml., of which 0·2 mg. was 'direct' and 1·1 mg. 'indirect'.

Further Investigations

The patient's blood group was O, Rh positive. The direct Coombs test was negative and abnormal antibodies were not detected. Cold agglutinins were present to a titre of 1 : 32. The direct and indirect Donath-Landsteiner tests were negative. Schumm's test was positive. Electrophoresis of the haemoglobin in veronal buffer (pH 8·6) showed a single component with the mobility of haemoglobin A, and sickling could not be demonstrated. Heinz bodies were not present. The osmotic fragility of the red cells was within normal limits. The V.D.R.L. test was negative.

The quantitative Ham's acid-serum test was positive, 23% haemolysis of patient's cells occurring in acidified normal serum. Haemolysis did not occur in unacidified or inactivated serum. Haemolysis by high-titre cold antibodies was 31%, there being no haemolysis of control cells. The Crosby test was positive, with a 50% increment in the amount of haemolysis in acidified serum with added thrombin, as compared with acidified serum without thrombin. Haemolysis of clotted blood left at 37°C. for 2 hours (heat-resistance test) was 12·5%, with no haemolysis of control blood. Red-cell acetylcholinesterase was 0·22 Δ pH per hour (normal range 0·65—0·95 Δ pH per hour). Alkaline-phosphatase activity could not be demonstrated in the patient's neutrophils, but was present in control smears.

A red-cell survival study (Fig. 1) showed a half chromium time ($T\frac{1}{2}{}^{51}Cr$) of 8 days (normal 30 ± 2 days) with mean cell life 28 days

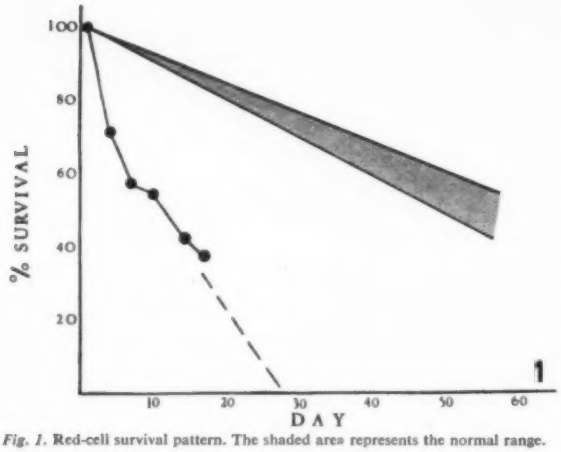

Fig. 1. Red-cell survival pattern. The shaded area represents the normal range.

(normal 100—130 days). Red-cell destruction was thus taking place at approximately 4 times the normal rate.

Course and Treatment

On 13 July, 2½ litres of blood were transfused. The haemoglobin value after transfusion was 11·3 G. per 100 ml., with 1,400 leucocytes per c.mm. (neutrophils 17%). Between 13 and 19 July the urine remained clear, and the patient was discharged from hospital. One week later she returned, complaining of the passage of dark-red urine. Physical examination showed pallor and mild jaundice. The haemoglobin value was now 6·3 G. per 100 ml., with reticulocytes 29% and leucocytes 2,100 per c.mm. (neutrophils 18%). Platelets were 50,000 per c.mm. Specimens of urine passed at intervals during the day varied in colour from dark red to almost black. Morning specimens were consistently blackish and those in the late afternoon varied from light to dark red.

On 2 September, a further blood transfusion was given, and the patient was subsequently discharged from hospital.

DISCUSSION

The findings in this patient can be described as classical of PNH. The haemoglobinuria was paroxysmal, and was more pronounced in the early morning specimens. The blood picture showed a haemolytic anaemia with reticulocytosis, intravascular haemolysis, and mild hyperbilirubinaemia, and the red-cell life-span was markedly diminished. There was leucopenia with persistent neutropenia, and thrombocytopenia, typical features of PNH. The various haemolytic tests were strongly positive. The red cells were deficient in the enzyme acetylcholinesterase, and deficiency to this degree is apparently specific for the disease.[5-7] Alkaline-phosphatase activity could not be demonstrated in the neutrophils, lack of this activity having being described in PNH by Beck and Valentine.[8] There was very marked haemosiderinuria, confirmed by chemical analysis.

PNH would be expected to occur in the Bantu, for the disease is well described in the American Negro. Crosby[9] referred to Negro patients, and 3 of the 6 patients described by Hartman and Auditore[10] were Negroes.

SUMMARY

A case of paroxysmal nocturnal haemoglobinuria in a Bantu female is described.

We wish to thank the Director, South African Institute for Medical Research, for facilities to carry out this study; Prof. J. V. Dacie for supplying the serum containing high-titre cold antibodies; Dr. V. H. Wilson for his help; Mr. D. Hart and Miss V. Brandt for technical assistance; and the Superintendent, Baragwanath Hospital, for permission to publish the case.

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THE GASTRIC FUNDUS

ITS PRESENTATION AS A 'MASS' BELOW THE LEFT DOME OF THE DIAPHRAGM

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The empty fundus of the stomach may sometimes produce a rounded shadow which is seen to lie below the left dome of the diaphragm in the plain radiograph of the abdomen.

Although this feature is probably well known, I am prompted to draw attention to it, having encountered instances in which the shadow has been misinterpreted and considered to be a cyst or tumour. Three illustrative cases are presented, as examples:

Case 1

(W.v.B.). Male, aged 40 years. A pyelogram taken during the investigation of his hypertension (Fig. 1) showed a 'mass' above the left kidney. The question of cyst or tumour was raised. However, barium in the gastric fundus confirmed the true nature of the shadow (Fig. 2).

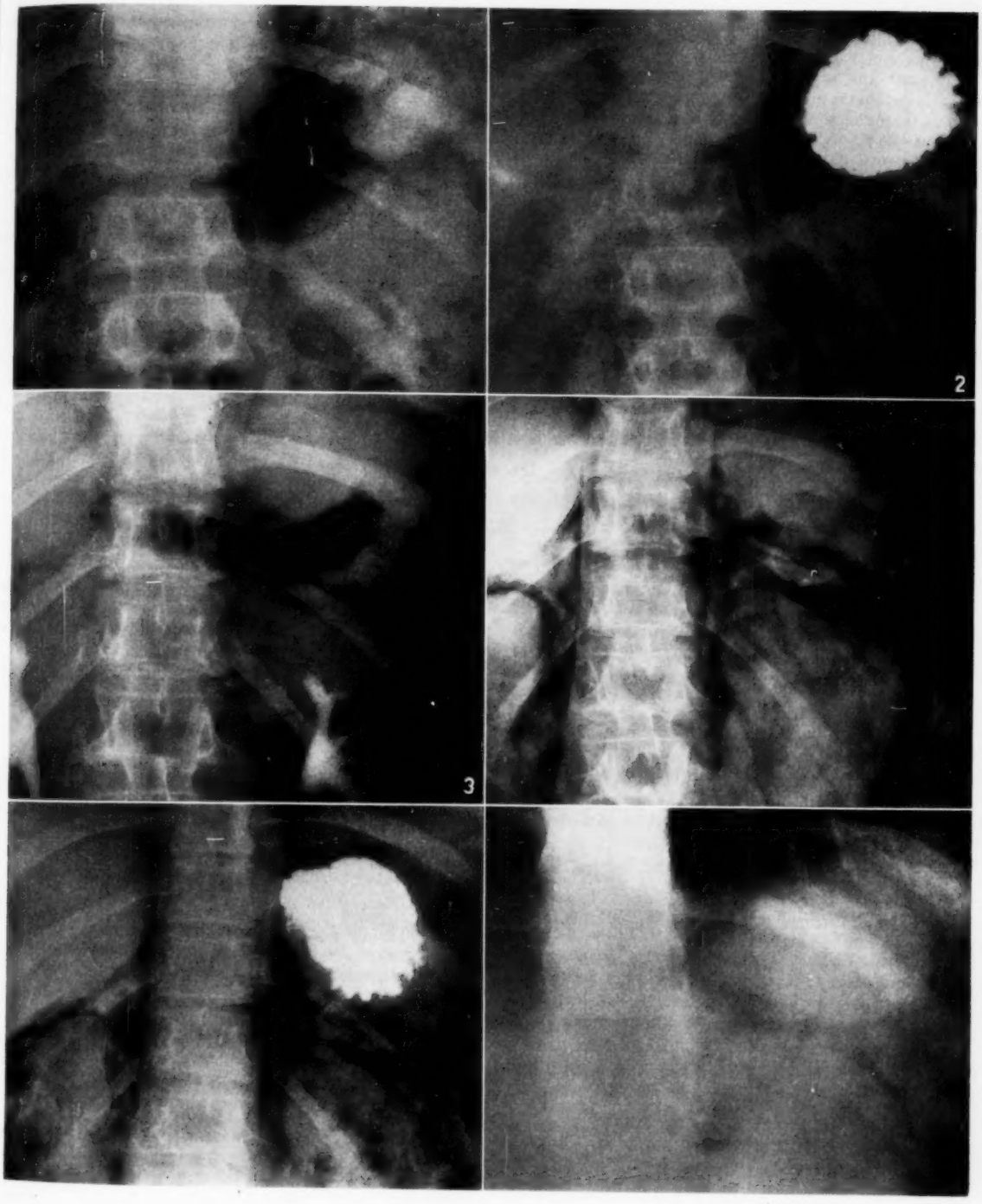
Case 2

(J.P.). Female, aged 23 years, with Cushing's disease. A rounded shadow is visible above the left kidney in the pyelogram (Fig. 3). Presacral air insufflation revealed no adrenal tumour, though bilateral adrenal hyperplasia was confirmed at operation. Note the shadow of the gastric fundus (Fig. 4) and the picture after ingestion of a small quantity of barium (Fig. 5).

Case 3

(L.W.). Male, aged 34 years. The patient was referred to a surgeon for a vague aching pain in the left renal angle. A plain radiograph of the abdomen revealed a 'mass' in the left upper quadrant (Fig. 6). The patient was a sheep farmer and a member of his family had suffered from hydatid disease. In view of this, it was considered probable that the 'mass' was a hydatid cyst. However, a laparotomy revealed no evidence of any abnormality in this region, and a subsequent radiograph showed no evidence of the oval density previously seen. The shadow is considered to be that of the gastric fundus.

Fig. 1. The rounded density of the gastric fundus is seen above the left kidney, contrasted against the air in the rest of the stomach (Case 1). *Fig. 2.* Barium in the fundus (Case 1). *Fig. 3.* Rounded shadow in left upper quadrant. Note relationship to rest of air-containing stomach (Case 2). *Fig. 4.* Same case after presacral air insufflation (Case 2). *Fig. 5.* Same case with barium in the fundus (Case 2). *Fig. 6.* Large oval opacity below left hemidiaphragm which was misinterpreted as a hydatid cyst (Case 3).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3

DISCUSSION

The appearance of the gastric fundus as a rounded shadow beneath the left dome of the diaphragm was described by Nathanson[1] and is discussed by Samuel,[2] who mentions that it is seen in the prone radiograph, though we have usually seen it in pyelogram films which are taken in the supine position. It occurs particularly in radiographs of hypersthenic subjects in whom the empty or slightly filled fundal loculus tends to hang downwards in the supine position. The fundus and the upper portion of the body of the stomach combine to produce this shadow.

The true nature of the 'mass' can easily be recognized by (1) its position, (2) its relationship to the rest of the stomach, which usually contains a small quantity of air, (3) its alteration in size and shape in serial films, (4) its alteration or disappearance in different radiographic positions, and (5) introduction of barium into the stomach.

SUMMARY

The empty gastric fundus sometimes produces a rounded shadow in the left upper quadrant in the plain radiograph of the abdomen.

The shadow may simulate a tumour or cyst.

The films of 3 cases are reproduced, illustrating the rounded shadow of the gastric fundus above the left kidney. Its true nature is confirmed by filling it with barium.

I wish to thank Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital, for permission to publish this report. I should also like to thank Dr. L. Werbeloff, Head of the Department of Radiodiagnosis, Groote Schuur Hospital, for his help, and Dr. E. G. Shulman, who first brought this feature to my notice.

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SALMONELLA WOODSTOCK

'N NUWE SEROTIPE [16: Z_{42}:1, (5), 7]

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Op 9 Junie 1960 het ons van 'n ontlastingsmonster 'n Gram-negatiewe kiem gekweek wat biochemies soos 'n salmonella gelyk het (ureum, negatief; dekstrose, suur en gas; mannitol, suur en gas; saccharose, negatief; laktose, negatief; H_2S, positief; indol, negatief; raffinose, negatief; inositol, negatief; dulsitol, suur en gas; sorbitol, suur en gas; eskulin, net suur; xylose, net suur).

Die pasiënt was 'n dame, 18 jaar oud, wat een jaar lank diaree gehad het en met die diagnose van verdagte amebiese disenterie toegelaat is.

Serologies was daar primêr net 'n sterk reaksie met die 0-agglutinien 16. Die beweeglike kiem was in die onspesifieke tweede fase teenwoordig, en het as sodanig met die agglutiniene 1, (5), en 7 gereageer. Dit was vir ons en ook vir die Salmonella-sentrum in Londen onmoontlik om die spesifieke eerste fase te verkry. Dr. P. R. Edwards (Sentrum vir Infektiewe Siektes, Chamblee, Georgia, V.S.A.) was die eerste om daarin te slaag om die spesifieke eerste fase te bepaal en gevolglik die volledige formule van hierdie kiem daar te stel. Die eerste fase-sweephaar-antigeen is z_{42}, en die hele formule is derhalwe: 16: z_{42}: 1, (5), 7.

Aangesien die pasiënt die infeksie waarskynlik in die Kaapse voorstad, Woodstock, opgedoen het, het ons aan hierdie kiem die naam *Salmonella woodstock* gegee.

S. woodstock was primêr weerstandig teen penisillien, streptomisien, eritromisien en novobiosien, maar gevoelig vir chloramfenikol, kanamisien, neomisien, en alle tetrasiklienes.

SUMMARY

Salmonella woodstock, a new serotype [16: z_{42}: 1, (5), 7].

A Gram-negative germ, isolated from the faeces of an 18-year-old woman, with the suggestive diagnosis of amoebic dysentery, was found to be a new serological type of the salmonella group. (Chemical properties: urea, −; dextrose, acid and gas; mannitol, acid and gas; saccharose, −; lactose, −; H_2S, +++; indol, −; raffinose, −; inositol, −; dulcitol, acid and gas; sorbitol, acid and gas; aesculin, only acid; xylose, only acid.)

On serological examination *S. woodstock* was strongly agglutinated only by the 0-antiserum 16. The motile germ was primarily agglutinable only by phase-2 antisera. It was impossible, either for us or the Salmonella Reference Laboratory, London, to obtain the specific phase 1. Dr. P. R. Edwards (Communicable Disease Center, Chamblee, Georgia) succeeded in growing phase 1 and, therefore, in determining the definite sero-formula: 16: z_{42}: 1, (5), 7.

In view of the fact that the patient probably contracted the disease in Woodstock, we named the new type *Salmonella woodstock*.

S. woodstock was primarily resistant to penicillin, erythromycin, streptomycin and novobiocin. It was sensitive to chloromycetin, kanamycin, neomycin, and all the tetracyclines.

Ek wil my dank uitspreek teenoor my hele tegniese personeel. Graag wil ek ook Dr. Joan Taylor van Londen, Prof. F. Kauffmann van Kopenhagen, en Dr. P. R. Edwards van Georgia bedank vir die bevestiging van die nuwe ontdekking.

Verder ook my dank aan die Mediese Superintendent van die Karl Bremer-hospitaal vir toestemming tot hierdie publikasie.

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE BEHOEFTE AAN PROFESSIONELE PERSONE

Een van die kenmerke van 'n beskaafde gemeenskap is die feit dat hy 'n hoë persentasie van opgeleide persone het wat spesiale skoling ontvang het om 'n ‚geleerde professie' te beoefen. En dit is nou juis op hierdie gebied waar ons meen dat ons die waarskuwende ‚skrif teen die muur' sien.

Een van die mees verontrustende tekens van die tyd waarin ons leef, is naamlik die groot tekort wat daar in byna elke vertakking van ons lewe bestaan aan opgeleide en geskoolde professionele persone—vernaamlik, en dit is waaroor ons hier meer besonderlik gemoeid is, op die breë terrein van maatskaplike en opvoedkundige dienste.

Om mee te begin is daar 'n baie groot tekort aan onderwysers, veral aan onderwysers van sekere vakke. Die gevolg is dat ons nie die soort van fondament kan lê by ons kinders wat ons graag sou wou sien nie. Daar is verder 'n groot behoefte aan meer maatskaplike werksters; en die menigvuldige probleme van aanpassing en heraanpassing in ons samelewing, wat reeds in die teken van spanning en drukte staan, kan nie bevredigend gehanteer word sonder die hulp van ervare maatskaplike werksters nie.

Met die ernstige tekort aan verpleegsters is ons almal bekend. Daar is hospitale waar verreweg die meerderheid van die verplegingspersoneel bestaan uit tydelike en wisselende persone. Ons is weliswaar baie dank verskuldig aan die lede van hierdie tydelike personeel, baie waarvan getroude vrouens is wat onder moeilike omstandighede werk; maar die toestand van sake is nogtans nie bevredigend nie. Ons het nie genoegsame bronne waaruit ons ons leiers en administrateurs onder die verpleegsters kan trek nie.

Ook in die mediese professie is daar die probleem van 'n veelkantige tekort wat ons in die gesig staar. Die feit van die relatiewe wanverdeling van en absolute tekort aan dokters (in terme van alle lede van die bevolking in al die gebiede van ons land), is al herhaaldelik genoem en beklemtoon in die mediese pers sowel as in die algemene pers.

Omdat daar in sekere stedelike gebiede 'n opeenhoping van dokters is, wil dit by oppervlakkige beskouing voorkom of ons genoeg of te veel dokters het. In terme van die totale bevolking van ons land, blank sowel as nie-blank; in terme van al ons landstreke en die aantal dokters wat beskikbaar is in afgeleë dele van die land; en in terme van die bevolkingsaanwas oor die algemeen, het ons egter 'n ernstige tekort op hierdie gebied.

Deur die saak so te stel soos ons nou net gedoen het, kom ons natuurlik dadelik te staan, nie net voor die probleem van 'n absolute tekort nie, maar ook voor die probleem van 'n geografiese wanverdeling wat 'n relatiewe tekort skep.

Die probleem van die tekort aan dokters het baie fasette. Ons het alreeds na een faset van die probleem verwys—die wanverdeling van dokters tussen die platteland en die stedelike gebiede. 'n Ander aspek van die probleem is die feit dat die tekort aan locums al chronies geword het. Daar is net nie genoeg persone om die noodsaaklike aflosdienste te verrig nie, en die dienste van diegene wat daar wel is, word onbetaalbaar duur. Die gevolg is dat dokters soms noodgedwonge hul praktyke en pasiënte verwaarloos, òf omdat hulle nie alles kan behartig nie, òf omdat hulle moeg en oorwerk voel.

'n Verdere faset van die probleem van die tekort aan dokters, waaroor ons skryf, is die feit en verwagting dat hierdie tekort, gemeet in terme van ons groeiende bevolking, stelselmatig groter word en nie kleiner nie. Die bestaande mediese skole en opleidingsfasiliteite kan, volgens berekening, nie genoeg gekwalifiseerde dokters lewer om die tekort uit te wis nie. Indien ons dus iets daadwerkliks wil doen om die vlak van ons lands- en volksgesondheid hoog te hou, moet ons ons opleidingsgeriewe radikaal uitbrei.

Die Universiteit van Stellenbosch tree as jongste mediese skool een van die dae te voorskyn met sy eerste gegradueerdes. Daar moet egter gedink word aan die stigting van nuwe opleidingsgeriewe op plekke soos Bloemfontein en Port-Elizabeth, en aan addisionele opleidingsgeriewe in Durban.

Tegelykertyd sal ons egter ook iets moet doen om te verhoed dat daar nie 'n groter opeenhoping van dokters in die stedelike gebiede plaasvind as wat daar alreeds is nie. Ons sal, met ander woorde, 'n oplossing moet vind vir die probleem van die relatiewe wanverdeling van dokters tussen stad en platteland.

Ook sal ons moet toesien dat die normale en gesonde balans tussen algemene praktisyns en spesialiste nie verder versteur word nie. Op die oomblik is daar 'n sterk neiging vir hierdie balans om ongunstig te verloop. Gedurende die vorige jaar was daar, byvoorbeeld, 'n vermeerdering van minder as 1% van die aantal algemene praktisyns in die land, teenoor 'n vermeerdering van ongeveer 6% van die aantal spesialiste.

Om al hierdie probleme, en baie ander wat ons nie genoem het nie, die hoof te bied, sal ons ons beste vermoëns moet mobiliseer en inspan. As 'n samelewing sal ons alles in ons vermoë moet doen om ons jongmense beroepsbewus en veral professiebewus te maak. Ons sal moet besef dat dit noodsaaklik is vir 'n volk om die geleerdheid van sy jongmense as 'n beleggingsbron te gebruik. En ons sal moet besef dat dit veral die professies is wat in die teken van maatskaplike dienste staan, wat 'n deurslaggewende bydrae sal kan maak tot die skat van menslike geluk en welsyn in die toekoms.

As lede van die mediese professie sal ons ook ons bydrae moet maak. Dit wil vir ons voorkom of daar op hierdie gebied 'n waardige en verhewe doel vir die Mediese Vereniging en sy bestuursliggaam, die Federale Raad, wag. Stemme het reeds al opgegaan wat kla oor die feit dat die Federale Raad so 'n groot deel van sy tyd bestee aan besprekings van slegs 'n ekonomiese aard. Laat ons die eer en belange van die mediese beroep, en dus uiteindelik ook van die pasiënte wat ons dien, op die hoogste vlak behartig deur die uitdaging van hierdie groot probleem in die gees van persoonlike verantwoordelikheid te aanvaar. Laat ons dus aan die wêreld toon dat ons bewus is van hierdie groot kulturele en beskawingstaak wat op ons wag.

ATHLETES

There is a widespread belief among most athletes and their instructors that there are some very special nutritional factors involved in the training and coaching and preparation generally of athletes for their contests. However, it seems to be established, on the basis of proper objective scientific study, that any well-balanced diet is all that athletes require for the best possible performance.[1]

Primitive tribes have their superstitions based on the idea that certain foods, such as the meat of certain animals, endow the consumer with strength, endurance, and courage. The eating of large quantities of meat has been advocated by trainers for athletes under their care to replenish muscle substance supposedly lost during severe exercise. It is obviously difficult to evaluate the effect of food components on physical performance, since so many factors and variables are involved, such as motivation, differences in the conditions and types of work performed, and the wide range of individual performance. The observed effects are often essentially psychological, the athlete doing better because he expected to perform better; or they may result from progressive training and the improvement gained thereby. Well-designed and well-controlled experiments are very important. Laboratory studies are not the same as those conducted on the athletic field, and in sport there are different types of stress imposed upon the body. The types of stress that arise from tennis are, for instance, quite different from those that arise from field events, such as high-jumps or sprints, mile runs, or marathons.

One of the most important considerations in the nutrition of an athlete is the calorie cost. Depending on the daily exercise done, the calorie requirement may be anything from 3,000 to more than 6,000 calories. A physically active individual may rely on his appetite as a guide to the intake of calories necessary to meet expenditure, but the problem is not simple. There are certain sports, for example wrestling, where a degree of overweight may be an advantage. However, for most activities overweight is a disadvantage, and some degree of calorie restriction may be needed. In younger athletes increased allowances should be made to meet the requirements of growth.

The influence of spacing meals and the number of meals has also been studied. Here again there are various opinions, but experiments suggest that athletes should have three meals a day or perhaps more frequent lighter meals, particularly if the exercise practised is of long duration. Ingestion of sugar before light exercise or intense exercise of short duration produces no demonstrable beneficial effect, but carbohydrate stores may become a limiting factor in prolonged events, and in such instances there is a case to be made for the ingestion of some form of sugar which is easily metabolized by the body. There is some evidence that in the cold the maintenance of thermal balance may be easier on a diet rich in fat. Although it has been repeatedly demonstrated that nitrogen excretion does not rise significantly after muscular work, there is a belief that a liberal protein intake is necessary during training. Certainly during periods of growth the requirements for high-quality protein are increased. Also, in most types of training, athletes will develop an increase in muscle mass for which additional protein will be required for tissue building. Normally, the protein requirements will be met by well-balanced meals. Wide variations of protein intake do not appear to influence performance capacity.

With regard to vitamins, no additional beneficial results seem to be provided by supplementing adequate diets, but there is insufficient evidence to rule out the possibility of increased demands caused by hard muscular work. Adequate intake of vitamins and other nutrients will be provided by well-balanced meals.

Other food substances of special nature have produced no improvement in performance. Caffeine-containing beverages may have an effect in decreasing awareness of fatigue, but large amounts are not to be recommended. As far as salt is concerned, use of any amount greater than that generally contained in a meal appears to be necessary only in unusually hot climatic conditions.

The optimum diet for an athlete in training is therefore generally not essentially different from that of any normal individual. The diet should provide for individual needs in respect of growth, maintenance, and superimposed activity. Undernutrition and malnutrition impair performance, and the nutritional status of the athlete should be assessed before the training season so that the diet may be planned according to the specific need for weight gain or weight loss.

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CHARACTERISTICS OF AGEING — CAPE JEWISH AGED HOME AS AN EXAMPLE

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People differ from each other as a result of the interaction of diverse genetic causes and environmental factors. Hereditary effects are more influential in shaping the young, but with the passing of years, nurture and general ecological conditions gradually assume a dominant rôle. The factors involved are many, great, and complex and, after a time, overshadow genetic effects.

Hence, as age advances and the individual's mobility decreases progressively, while his dependence increases at the same pace, the effects of earlier varying or digressing factors cancel each other. Insidiously, but relentlessly, the order of their development or involution becomes almost uniform. Homogeneity, like monotony, overtakes all at varying speeds.

Accordingly, in this paper, an assumption is made that data obtained from the analysis of a small, random number of elderly individuals may be accepted as an approximate index of the composition, likely prognosis, trends of development and needs of larger groups of ageing people.

Though, at the time of this survey, there were less than 200 residents at the Cape Jewish Aged Home, they complied with the requirements of a random sample. Except for victims of communicable disease or serious mental impairment, almost

all handicapped Jewish applicants are accepted. There is no means test, nor is country of origin or language an obstacle. This sample, therefore, provides an index of the problems created by an ageing population and may constitute a guide towards future planning.

On 18 August 1959, the population of the Cape Jewish Aged Home consisted of 179 residents (exclusive of staff). Of these, 111 or 62·02% were females and 68 or 37·98% were males. The preponderance of females in the ageing population is a world-wide phenomenon. Of 200 candidates seeking admission to the Witwatersrand Jewish Aged Home from November 1958 to March 1960, 64% were females and 36% were males.[1] Going further afield, Franz Goldman,[2] in his survey of the residents of 5 Jewish Homes for the Aged in North America, found that of 530 residents, 330 were females and 200 males, the percentages being 62·3% and 37·7% respectively.

This is not an exclusively Jewish trait. In the state of Daghestan (USSR), Alikishiev[3] found that 200 persons more than 100 years old were divided into 123 women (61·5%) and 77 men (38·5%). Of between 600 and 700 centenarians in France, there is a considerably higher number of females than males.[4] Longevity in the female, compared with the male, is not confined to the human race, but is seen in various species of animals from mammals down to house flies.

AGE COMPOSITION OF THE RESIDENTS

Table I classifies the residents according to their calendar or chronological age. The declared age given by the resident or by his or her guardian was accepted as the actual chronological age.

TABLE I. CLASSIFICATION OF RESIDENTS OF CAPE JEWISH AGED HOME ACCORDING TO THEIR CHRONOLOGICAL AGE

Age (*years*)	*Females*	*Males*	*Total*	
			No.	%
Under 50	3	0	3	1·7
50—54	3	2	5	2·8
55—59	4	3	7	3·9
60—64	6	3	9	5·0
65—69	6	3	9	5·0
70—74	19	16	35	19·6
75—79	29	16	45	25·2
80—84	28	15	43	24·0
85—89	10	6	16	8·9
90—94	3	4	7	3·9
Total	111	68	179	100·0

This age composition is not peculiar to the Cape Jewish Aged Home. Statistics obtained at similar institutions in other countries reveal an approximately similar age distribution.

TABLE II. PERCENTAGE DISTRIBUTION OF AGES OF RESIDENTS IN THE CAPE JEWISH AGED HOME COMPARED WITH 5 NORTH AMERICAN HOMES AND THE WITWATERSRAND JEWISH AGED HOME

Age (*years*)	*Cape Jewish Aged Home* %	*5 North American Aged Homes* %	*Witwatersrand Jewish Aged Home* %
Under 60	8·4	0·4	6
60—64	5·0		
65—69	5·0	6·2	27
70—74	19·6	13·4	
75—79	25·2	26·4	
80—84	24·0	28·3	
85—89	8·9	17·9	67
90—94	3·9	4·9	
95—99	0	1·3	
100 and over	0	0·4	
Age not recorded	0	0·8	0

The residents of Jewish Aged Homes in North America are older than those of Jewish Aged Homes in South Africa, as can be seen from Table II.

Thus at 5 North American Aged Homes about 6·6% of the residents are under 70, and about 92·6% are over 70 years of age, in contrast to 18·4% under 70 and 81·6% over 70 years old at the Cape Jewish Aged Home, and 33% under 70 and 67% over 70 at the Witwatersrand Jewish Aged Home.

DISEASES OF RESIDENTS AT THE CAPE JEWISH AGED HOME

Diagnosis is based mainly on symptoms and signs detected by the general practitioner in charge of the patient. Where a satisfactory diagnosis cannot be made by the general practitioner, ancillary methods of investigation are utilized or a consultant is called in.

Since the number of patients is small, the 56 pathological conditions occurring at the Home are reduced to 12 groups. Diseases are placed together in one group, not necessarily because of their affecting the same organ system or having a similar pathology, but because of the similarity of effect or disability they produce. To give an extreme example: Hernias and fractures are placed in one group because the handicap of both is mechanical. A patient suffering from more than one disease in any one system is counted once only, e.g. a sufferer from coronary heart disease and hypertension appears once only in the cardiovascular group.

On the other hand, a patient suffering from hypertension and an affection of the prostate appears under both the cardiovascular and genito-urinary groups. Residents are thus classified into the following groups:

1. *Cardiovascular* — arteriosclerosis, atherosclerosis, coronary disease, peripheral gangrene, heartblock, hemiplegia, hypertension, peripheral vascular disease and Raynaud's disease.
2. *Mental impairment* — alcoholism, dementia praecox, difficult to control, depressions, neuroses, paranoia, psychoses, schizophrenia, senile dementia, and minor psychological upsets.
3. *Metabolic* — cholecystitis, cirrhosis, diabetes mellitus, gout, myxoedema, obesity, and osteoporosis.
4. *Nervous* — aphasia, epilepsy, parkinsonism and spasticity.
5. *Genito-urinary* — gynaecological, prostatism, urological diseases (including incontinence).
6. *Special senses* — blepharitis, blindness, handicapping defective vision, deafness, dumbness, glaucoma, tinnitus.
7. *Rheumatic* — arthritis, osteoarthritis, rheumatoid arthritis.
8. *Mechanical* — amputations, fractures, and hernias.
9. *Gastro-intestinal* — diseases affecting the gastro-intestinal tract, excluding hernias.
10. *Cancer.*
11. *Respiratory* — asthma, pulmonary diseases, bronchial disorders.
12. *Skin diseases.*

Table III places diseases encountered at the Cape Jewish Aged Home in descending order of appearance according to the above-mentioned groups and according to sex.

It may be valuable to examine a few of the most prominent

TABLE III. DISEASES ENCOUNTERED AT THE CAPE JEWISH AGED HOME IN THE WHOLE POPULATION OF THE HOME AND IN THE FEMALES AND MALES SEPARATELY

Disease group	*Total population*		*Females*		*Males*	
	No.	*%*	*No.*	*%*	*No.*	*%*
1. Cardiovascular	71	40·0	43	38·7	28	41·1
2. Mental impairment	55	31·0	39	35·1	16	23·5
3. Metabolic	28	25·6	21	18·9	7	10·3
4. Nervous	26	14·5	17	15·3	9	13·1
5. Genito-urinary	25	13·9	8	7·2	17	25·0
6. Special senses	22	12·3	14	12·6	8	11·7
7. Rheumatic	21	11·7	16	14·4	5	7·3
8. Mechanical	14	7·7	9	8·1	5	7·3
9. Gastro-intestinal	13	7·3	7	6·3	6	8·8
10. Cancer	7	3·9	3	2·7	4	5·9
11. Respiratory	6	3·3	6	5·4	0	0·0
12. Skin	3	1·7	3	2·7	0	0·0

diseases in greater detail, and to study their age and sex distributions.

Cardiovascular Diseases

Cardiovascular disease is an all-pervasive condition in elderly persons. Even old people who lack complaints are often found to be affected. Thus, it was reported from the Chicago Medical School[5] that of 500 people, 50 years old and over, 60% had cardiac enlargement, 42% had murmurs, 22% had hypertension, 30% had inverted T waves and 6% showed cardiographic evidence of old infarcts. For practical purposes the cardiovascular system may be regarded as the principal factor affecting human health and longevity. Its deteriorations, whether showing themselves as cardiac malfunction or degeneration of the arterial tree, are the main cause of morbidity and mortality in the elderly. Hence cardiovascular disease constitutes the greatest challenge to the medical profession.

Table IV shows that it is the principal cause of disease at the Cape Jewish Aged Home. Seventy-one residents, of whom 43 are females and 28 are males (39·6% of the total population, 38·7% of the females and 41·2% of the males), suffer from cardiovascular disease; it also shows that males are slightly more liable to these affections than females, and that the age group 70 - 79 is most affected in both sexes.

TABLE IV. SUFFERERS FROM CARDIOVASCULAR DISEASE CLASSIFIED ACCORDING TO AGE GROUPS AMONG THE TOTAL POPULATION OF THE HOME AND THE FEMALES AND MALES SEPARATELY

Age group	*Total population*		*Females*	*Males*
	No.	*% of age group*		
50—59	3	25·0	1	2
60—69	7	38·8	5	2
70—79	37	46·25	21	16
80—89	22	37·3	15	7
90 and over	2	28·6	1	1
Total	71		43	28

The greater liability of men to cardiovascular disease is a universal phenomenon. As an example, an analysis of cardiovascular-renal mortality for the Chicago population, aged 25 - 64, in 1951 and 1953, showed that the death rate from arteriosclerotic heart disease was 'severalfold higher in men than women'.[6]

Cerebrovascular Accidents

Hemiplegia, usually resulting from cardiovascular disease, also shows a preponderance of males over females, and here, too, the age group 70 - 79 has proportionately most sufferers. Eighteen residents, or 10% of the total population, have or have had a stroke. Of these, 10 are females (9% of female residents) and 8 (11·8% of males) are males. This tallies with the findings of others; of 260 patients with cerebrovascular accidents, Greppi[7] found that 65% were males and only 35% females, in spite of the fact that the number of aged women in that group was greater than that of aged men. Unfortunately the exact proportion of males to females in the patients investigated is not stated. In his group the greatest number of hemiplegias occurred in persons between the ages of 60 and 70.

Mental Impairment

As used in this paper, this term is an omnibus designation. Included in the group 'mental impairment' are 36 patients with the following disabilities: offensive eccentricity, abnormal irritability, neuroses, early psychoses, depressions, and chronic uncooperation. There are also 2 alcoholics, 1 patient with dementia praecox, 2 with paranoia, and 24 with senile dementia.

The magnitude of this problem the world over can be gauged from the statement that 9 million Americans are victims of serious mental disorders and that half the space in American hospitals is occupied by mental patients. As a counterweight, the USA devotes 30 million dollars annually to psychiatric research. It is a huge sum, but fades into insignificance when compared with the 300 million dollars which is spent annually in the USA on chewing gum.[8]

In Britain the position is no better.[9] There it is estimated that one out of every hundred children is mentally deficient.

Mental impairment of the elderly arises from a multiplicity of causes, and shows itself in an equally great number of forms, all of which are uniform in that they destroy the individual's self-sufficiency and the harmony of his residence.

In an aged institution, there is a progressive increase in those with mental impairment, since many whose minds are healthy on admission deteriorate as they grow older. It thus constitutes one of the greatest perils menacing persons of advancing years; fortunately, however, the alert clinician, not dominated by an attitude of fatalism, may often uncover preventable or curable causes. That abnormal behaviour often masks systemic disease is not sufficiently well realized.

The Cape Jewish Aged Home claims, with justifiable pride, that the mentality of many a person who, on admission, displayed some mental aberration, has improved after a short stay at the Home as a result of understanding care, amelioration of some physical ailment, sensible feeding and controlled medication. Thus, a number were saved from being committed to mental institutions, and their relatives from an unjustified stigma. Fifty-five residents, corresponding to 30·7% of the

TABLE V. SUFFERERS FROM MENTAL IMPAIRMENT CLASSIFIED ACCORDING TO AGE GROUPS AMONG THE TOTAL POPULATION OF THE HOME AND THE FEMALES AND MALES SEPARATELY

Age group	*Total population*		*Females*		*Males*	
	No.	*% of age group*	*No.*	*% of age group*	*No.*	*% of age group*
Under 50	2	1·1	2	66·6	0	0·0
50—59	7	58·3	3	42·8	4	80·0
60—69	8	44·4	6	50·0	2	33·3
70—79	19	23·8	15	31·2	4	12·5
80—89	18	30·5	12	31·6	6	28·6
90 and over	1	14·2	1	33·3	0	0·0
Total	55		39		16	

total, suffer from mental impairment. Of these, 39 are females (35·1% of the females) and 16 are males (23·5% of the males) —Table v.

The majority of residents under 65 are admitted because of mental frailty, hence the high percentage of mental impairment among them. Females thus appear to be more liable to mental affections than males. Perhaps a compensation for the cardiovascular position!

The percentage of those who deviate from the mental norm is not extraordinarily high; similar percentages are found in other institutions for the aged. Thus, the percentage of 'mentally confused' in 5 North American Jewish Aged Homes is given as 42·1%.[10]

A similar condition prevailed in 1952 at the Home for Aged Hebrews in New York, where 16·8% of the residents suffered from mental disorders (18·3% females, 13·9% males).[11]

Diabetes

The prevalence of glycosuria is not as high at the Cape Jewish Aged Home as at some institutions in the USA. Here, 18 residents, or 10% of the total population, have glycosuria. At the Montefiore Home, Cleveland,[12] 29 out of 193, or 15%, are described as diabetics, while R. L. Brandt[13] reported at the Fifth International Gerontological Congress in August 1960, that of 120 inmates investigated at Michigan, a presumptive diagnosis of diabetes mellitus could be made in 40.

At the Cape Jewish Aged Home, patients with a fasting blood sugar of over 120 mg. per 100 ml. are classified as diabetics, therefore renal glycosuric patients are not included in this group. On the other hand, since the blood sugar of persons who do not show glycosuria is seldom examined, the possibility of some diabetics escaping detection cannot be ruled out.

Of the 18 diabetics, 14 are females (12·6% of the female population) and 4 are males (5·9% of the males). This does not agree with Osler's *Principles and Practice of Medicine* (1924) which states that 'men are more frequently affected than women, the ratio being about three to two'.

At the Witwatersrand Jewish Aged Home, 13% of the females and 10% of the males suffer from diabetes.[1] One third of our diabetics suffer from cardiovascular disease, and an equal number are mentally impaired. Apparently the detectable incidence of diabetics is changing, more females now being liable to it than males. This is no racial peculiarity. In a study of 250 urbanized Africans, the peak incidence was in the sixth decade, and the disease was commoner in females in the proportion of 1·6:1.[14]

FUNCTIONAL CAPACITY OF THE RESIDENTS

Information obtained from grouping residents of a Home into their age or defect components is clinically useful and academically desirable, but is an inadequate guide for enabling the management to assess the type and amount of medical and custodial staff and equipment it needs to provide. To decide on this, the extent of each individual's dependence on the assistance of others is required. This can be deduced from measuring or assessing the person's ability to cope with his physical needs and his mental and social problems.

The residents of the Home were therefore divided into three functional-capacity classes according to their compliance with the following criteria, taking into consideration the individual's own estimate of his health, something which influences his effectiveness to a considerable extent:

Class 1 (*Fit*)

An ambulant individual rated by himself and others as in moderately good physical and mental health and able to cope with the ordinary situations of everyday life. He is therefore able to look after himself, and is a resident of the Home because of pecuniary or social circumstances. He may have signs of diminution of reserve and slowing down of his faculties or he may harbour a progressive pathological condition, but the symptoms and signs of these have not yet reached the clinical threshold.

Class 2 (*Moderately Fit*)

(*a*) An ambulant individual capable of coping with ordinary problems of life, though suffering from a clinically discernible disease. He requires periodic attendance by a doctor and/or nurse, but is otherwise capable of looking after himself.

(*b*) A patient convalescing from an acute disease, or an exacerbation of a chronic condition.

Class 3 (*Unfit*)

A person suffering from one or more diseases necessitating frequent control by a doctor or nurse, e.g. (*a*) a bed- or chair-fast patient, (*b*) a person incapable of attending to his/her personal hygiene, (*c*) a harmless mentally disordered patient, or (*d*) a terminal patient.

It was found that 45 individuals or 25·1% of all the residents belonged to Class 1, 39 individuals or 21·8% of the residents belonged to Class 2, and 95 individuals or 53·1% belonged to Class 3.

Thus about 75% of the residents require frequent or periodic medical and/or nursing supervision. The preponderance of hospital or nursing-home types of persons at the Cape Jewish Aged Home is in no way unique. It accords with the trend in other countries where the chronic sick are steadily ousting the healthy aged. Thus, in his 1959 annual report, President Affelder, of the Montefiore Home of Cleveland,[12] stated: 'Twenty-five years ago, at least 90% of our residents were well, aged persons: today less than 10% of our residents can be qualified as such'. Moreover, not much difference is now discernible in the health status of residents of some aged homes and of some chronic-disease hospitals. In 1959, Baltimore (USA) City Chronic Disease Hospital had under its care 230 medical patients. Of these only 42% required constant medical and nursing care.[15]

For this reason some social and medical scientists are advocating that moderately healthy, self-sufficient aged persons should not reside at Aged Homes, but should be accommodated in apartments or flats or boarding houses specially designed for such people or should be boarded out with private families under the surveillance of a supervising organization. The present Aged Homes, on the other hand, should be equipped with adequate facilities for diagnosis and treatment of chronic diseases, and should be reserved only for victims of long-term diseases or crippling disabilities. It would appear that the Cape Jewish Aged Home is moving in this direction.

Drs. Franz Goldman and Neva Deardorff,[16] after reviewing the position in the USA, recommended a modification of the above scheme. They stated: 'Instead of drawing a line of demarcation between homes for aged people in apparent good health and homes for aged people designed to serve the sick as well as the healthy, the emphasis should be placed on

development of (1) apartments for oldsters who can take care of themselves or need only some help, and (2) good institutions for long-term care of chronically ill or seriously disabled patients *regardless of age*. These two types of physical facilities should be in close proximity'.

WHICH IS THE HEALTHIER SEX?

At the Cape Jewish Aged Home, males are healthier than females, as is demonstrated in Table VI, which analyses the males, females and total population separately into the three functional-capacity classes. It will be seen that the percentage of unfit persons is significantly higher among the females than the males.

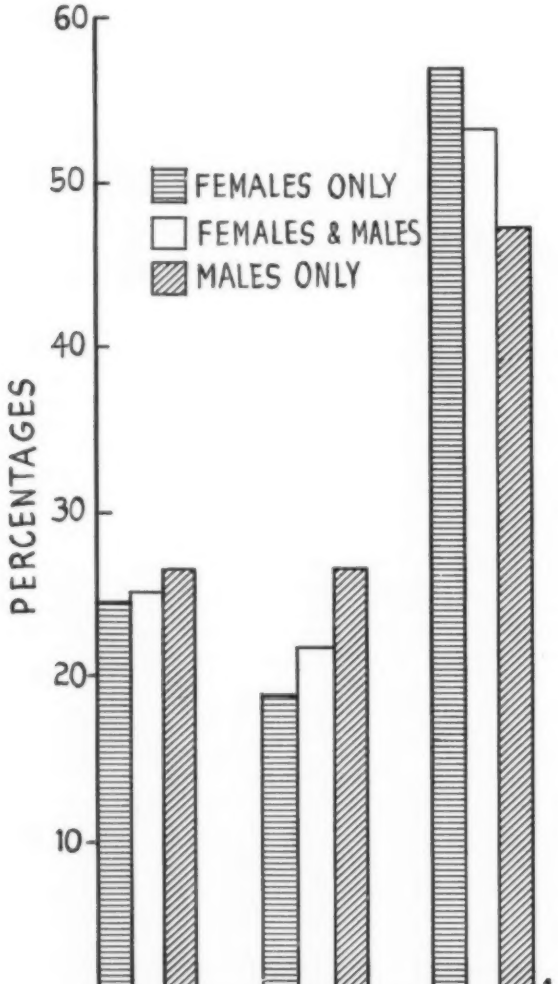

Fig. 1. See text.

TABLE VI. THE THREE FUNCTIONAL-CAPACITY CLASSES CLASSIFIED ACCORDING TO THE TOTAL POPULATION OF THE HOME AND THE FEMALES AND MALES SEPARATELY

Class	*Total population*		*Females*		*Males*	
	No.	*%*	*No.*	*%*	*No.*	*%*
1	45	25·1	27	24·3	18	26·5
2	39	21·8	21	18·9	18	26·5
3	95	53·1	63	56·8	32	47·0
Total	179	100	111	100	68	100

The histograms in Fig. 1 compare graphically the relative numbers of men and women in each capacity class, and illustrate that the percentage of relatively healthy men is greater than that of women.

It may be argued that these figures are of a specially selected group and that a higher percentage of fit males than fit females (or to put it differently, a higher percentage of unfit females than males) seek admission to the Home. Unfortunately the residents were not classified into functional-capacity classes on admission, therefore no data to verify or disprove this contention are available at the moment.

However, other workers have produced evidence showing that men who reach old age are healthier than women. Thus, Pemberton[17] analysed the state of health of 284 elderly women and 192 elderly men not living in institutions, and found that 23·6% of females and 26·2% of males were fit; interestingly near to the percentages at the Cape Jewish Aged Home (Table VI).

Women live longer than men, but men are compensated by their suffering less ill health during their relatively shorter life.

SUMMARY

1. The ageing process follows a similar pattern in all countries with a Western mode of life.

2. Females are longer-lived than males, but in ageing are subject to more illness.

3. Cardiovascular disease and mental impairment are the predominant and gravest incapacitating illnesses of the aged.

4. To substantiate these conclusions, a survey is presented of the residents of the Cape Jewish Aged Home by sex, age, disease and functional capacity. It is compared with surveys elsewhere.

5. Some new trends in ageing and the housing of the aged are discussed.

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THE TREATMENT OF MILK IN INFANTILE GASTRO-ENTERITIS

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'Curds and honey shall he eat . . . . .' *Isaiah*, ch. 7, v. 15.

The title of this paper is not a misnomer. Time was when the treatment of gastro-enteritis was mainly concerned with the alterations in the composition of milk feeds, the affected infant taking second place; but latterly, with the increase in understanding of the profound biochemical changes occurring in gastro-enteritis, attention is being concentrated on the affected infant, and the make-up of the formula is now of secondary interest, as is also the frequency and character of the stools. The quaint practices of earlier days are giving way to more exact and scientific handling of the affected infant. The era of the 'stool-gazers',[1] 'clinical soothsayers' and 'formula fiddlers'[2] is rapidly passing, but is not yet quite over, and it will be profitable to consider how it came about that in infantile gastro-enteritis treatment was directed to the milk rather than the infant, and how our ideas on this subject have changed.

MILK IN GASTRO-ENTERITIS

The spectre of Czerny's 'Milchnahrschaden' (milk injury)[3a] still haunts the portals of paediatric instruction. All too frequently cow's milk is looked on as the cause of diarrhoea—a concept still being accepted by students, nurses and residents. Any raw novice to paediatrics, when confronted with a child having diarrhoea, is pretty certain to order diluted feeds without knowing why this should, or should not, be done. 'Put him on clear fluids for 24 hours' is almost an axiom of paediatric therapy. Early paediatric teaching and internship training leaves hard-to-erase impressions in the trainee's mind. It is difficult to unlearn errors and to question accepted dogma. Nurses automatically give $\frac{1}{8}$- or $\frac{1}{4}$-strength feeds if a child passes a loose stool.[4] Even most of the 'intelligent' public 'know' that milk is bad for diarrhoea, and that the fat in the milk is especially noxious. When an infant has a few loose stools it is quite the rule for the mother to ask: 'Must I stop his milk?'

Time was when physicians not only fiddled and juggled about with the formula, but also changed it on the basis of the appearance of the stool. Today most physicians are content to deal with glucose or sugared water, $\frac{1}{4}$-, $\frac{1}{2}$-, or full-strength feeds; while some—a small minority—are even impatient with this conception, believing that milk modification and dilution make no material difference to the course of diarrhoea, and, if anything, infants given full feeds (plus adequate extra fluids) do better than those who suffer $\frac{1}{4}$, $\frac{1}{2}$, or full iatrogenic starvation.

Historical Survey

The ancient Babylonians[5] wrote on diarrhoea, and on the medications and incantations necessary for its cure. Cholera infantum was described by Areteus, and by Rhazes;[6] and Thomas Phaer indicted teething as its cause—a view first suggested by Hippocrates.[7]

Geronimo Soriano,[8] a Spanish physician, wrote a book on diseases of children (1600) in which he advised starvation treatment for diarrhoea. A century later Underwood[9] also substituted broth for milk when the bowels were loose. Now, while the period of the Renaissance was one of general enlightenment, there was little, if any, enlightenment in the field of infant feeding. The period was marked by a massive increase in hand (artificial) feeding and in bad wet-nursing. As a result, infantile gastro-enteritis was rife and deadly. It was common knowledge that infants who were breast-fed by their mothers seldom became ill with the 'watery gripes', but that the hand-fed infants rapidly succumbed to it. The blame was laid at the door of the common denominator—animal's milk—and correctly so; until recent years, with the widespread practice of pasteurization, animal's milk remained foul and filthy.

Consequently there were determined efforts to rear artificially fed infants on food other than cow's milk—which resulted in the common use of pap and panada: cereals cooked in milk-free fluids. The outcome of such attempts at infant rearing was also catastrophic. If it should have happened that a baby was being fed partially on animal's milk, and then developed diarrhoea, certainly the milk was removed, being considered poisonous, as indeed it probably was in so far as it might have been the vehicle for the spread of gastro-enteritis.

By the 1800's, when Western paediatrics entered the modern and rational phase, animal's milk was known to be associated with diarrhoea and was roundly condemned by the physicians of the day as being poisonous to infants having loose stools.

Why it should have been poisonous was not fully appreciated until 100 years later, and this period of a century marks the gradual evolution of thought on the hazards of cow's milk in gastro-enteritis. Initially the opinion was held that various chemical and physical constituents (curds) of the milk were responsible for producing diarrhoea, toxicosis, dyspepsia, athrepsia, decomposition, intolerance, indigestion, and intoxication. At the present time there are more substantiated views on the bacteriology and virology of diarrhoea, on the chemistry and osmolarity of cow's milk, and on body fluids and renal function.

Before 1900 it was milk *per se*, even breast milk, that was condemned as being poisonous in diarrhoea. Cheadle,[10] of the Westminster Hospital, had stated this, and Eustace Smith[11] had written: '. . . . the treatment consists mainly in the careful regulation of the food. Milk in such a case is an irritant which must be strictly forbidden'.

After the turn of the century there arose the view that it was not milk *per se* that was hazardous, but only certain constituents of it. Such theories had even been mooted before 1900, when Biedert[3b] wrote that the casein of milk might be dangerous in diarrhoea, though he also had his reservations about fat. Escherich,[3c] while engaged on monumental studies on intestinal bacteria, still toyed with the possibility of carbohydrate fermentation and protein putrefaction in the bowel as causes of diarrhoea. However, it was only after 1900 that a great impetus was given to bowel 'dyspepsias' by Czerny,[3a] who blamed fat; by Finkelstein,[3d] who incriminated carbohydrate and milk electrolytes, and by Rotch,[12] who, like Biedert, noted that cow's milk had more protein than breast milk, and decided that milk protein was at fault.

Diarrhoea, being thought to be of dietetic origin, was treated by dietetic means. If a physician considered that one or other milk factor was at fault, then such constituent was reduced or replaced by another. All too often, all the milk constituents were incriminated, so that milk intake was severely curtailed by gross and long-standing dilution. When there was no response to this treatment, numerous modifications of milk were tried, and, as Marriott[13] later stated, 'any success which attended such efforts was usually to be attributed to the accidental giving of a sufficient amount of food'.

The principal physicians who possessed the esoteric knowledge of the cult of milk modification—largely based on 'stool-gazing'—were the three 'high priests' of the 1900s, Czerny,[3a] Finkelstein,[3d] and Truby King.[14] King was such a devoted believer in the evil propensities of milk that he even forbade breast milk in the presence of diarrhoea, and his influence, like that of his cohorts, spanned the world, and is still readily discernible today—especially among the laity, for whom the whole subject of infant feeding is still befogged in mysticism. Fischer,[15] in America, wrote two volumes on paediatrics (1928) and devoted a whole chapter to 'milk poisoning' complete with a photograph of such a case (a child with undernutrition and dehydration). Even Griffith[16] (the first 'Mitchell-Nelson') in his two volumes, reserved a paragraph for 'acute milk poisoning', though mercifully there is no illustration of such a condition. In England, Eric Pritchard was content to give no food whatever for 24 hours. He knew of the work of DuBois and Coleman (1912) which showed that in typhoid enteritis digestion and assimilation of food was almost normal, yet he wrote: 'I am quite unconvinced . . . and I still pin my faith to the method of low caloric feeding . . .'.[17]

Protein Milk

In keeping with his views that diarrhoea was caused by excessive fermentation of sugars within the bowel, Finkelstein[3d] devised his famous 'protein milk' (albumin milk, Eiweissmilch) in which the sugar was especially low, as was also the content of sodium and potassium. He reasoned that the fatty acids in the digested milk would then combine with the insoluble electrolytes (calcium and

magnesium) to form insoluble soaps and thus give bulk to the stools.[16] Protein milk was poorly named, for the percentage of protein in it was only 3% (fats 2·5%, sugar 1·5%, salts 0·5%) and the caloric value about 14 calories per oz. Despite a most complicated method of preparation, it became popular in America and was widely used for two decades; it is still in extensive use in southern Ontario.

Grulee,[19] Hill,[20] and Fischer[15] were most excited about the virtues of protein milk, and Brennemann[21] was lavish in his praise, as was Holt senior.[22] 'Casein is therefore indicated to combat diarrhoea', wrote Fischer, 'this teaching . . . reverses our former theories . . .'.[15] Grulee said: 'In decomposition . . . no artificial food can compare . . . with Finkelstein's and Meyer's albumin milk.'[19]

However, Morse[23] was not too enthusiastic about protein milk, and in England it did not become popular. Pritchard[17] heaped scorn on it: 'Personally, I agree with no part of the argument, and I regard Eiweissmilch as a very clumsy and complicated method of providing a mixture of the desired properties'.

Modified Milk

Concurrent with the widespread use of protein milk, and following it, milk was also modified in other respects, largely as a consequence of Czerny's imprecations against fat. Skimmed milk came into common use, also lactic-acid milk, and humanized milks, in particular the SMA (Synthetic Milk Adapted) of Gerstenberger.[24a,b]

The use of one or other modified milk in the treatment of gastro-enteritis has persisted until now, and the well-known doctors who advised such milk modification are too numerous to list, but among those who have diluted milk, humanized milk, replaced its sugars with dextrins, defatted milk, acidified milk or otherwise altered and emasculated it, there must be mentioned such authoritative names as Rotch,[12] Morse,[23] Czerny,[24a] Finkelstein,[3d] Truby King,[14] Hill,[20] Hess,[25] Still,[26] Griffith,[16] Holt snr.,[22] Grulee,[19] Pritchard,[17] Spence and Miller,[27] Brennemann,[21] Watkins and Paterson,[27] Paterson and Newns,[28] Jeans and Marriott,[13] Sheldon,[29] Slobody,[30] Spock,[31] Darrow,[32] Cooke,[33b] and Finberg.[34]

Even the more modern among these have assiduously pursued the path of the earlier masters. In the 1941 edition of their book, Jeans and Marriott[13a] gave consideration to the part played by fermentation of sugar in the causation of diarrhoea. In the 1947 edition[13b] this discussion was omitted, and a note was made that viruses may be responsible for epidemic diarrhoea of the newborn. In the 1955 edition of his book, Sheldon[29] wrote extensively on 'indigestion' and the various milk constituents which are alleged to cause such bowel disorders. The booklet 'Canadian Mother and Child', official publication of the Canadian Department of Health and Welfare[35] repeated the fiction about sugar and fat causing diarrhoea: 'If sugar is causing the trouble, definite signs and symptoms will appear. The baby will be restless, cry spasmodically, move its legs jerkily; usually the abdomen will be distended and tender to touch. The stools will be loose, frothy, sour smelling and irritating . . .'. 'If fat is at fault . . . the child may develop diarrhoea . . .'.[35]

The widely read and enormously influential Spock[31] advised that, in mild diarrhoea, diluted skimmed milk is the nutrient of choice, and that in serious diarrhoea all milk is forbidden for 24-72 hours 'depending on how soon the bowel movements improve in appearance'. (What an odd criterion for oral management!) 'Then proceed *very* gradually.' *The Complete Book of Mothercraft* agrees with this view.[36] Parsons and Barling,[27] Herweg *et al.*,[37] and Ebbs[38] repeat the usual advice about diluting milk and proceeding very slowly thereafter to full feeds. Mann *et al.*[39] deal extensively with the necessity for regrading of feeds and note that most patients take as long as 8-14 days to resume full feeds. Virtually all among the aforementioned stress the need for redilution of milk if there is a relapse of diarrhoea.

Darrow,[32] Cooke[33b] and also Finberg[34] advise prolonged dilution of feeds, but for different reasons entirely, reasons which will be considered presently.

Heresies

However, during the past half-century of the practice of milk modification in diarrhoea, there have been a number of heretics who have struggled against the flood of modified and diluted milk.

At the turn of the century, Pierre Constant Budin,[40] a remarkable Frenchman, noted that undiluted sterilized cow's milk markedly benefited infants with wasting and diarrhoea. Much later Hess[25] warned against prolonged initial starvation in the treatment of diarrhoea, and Jeans and Marriott,[13] while subscribing to the usual treatment by means of diluted skimmed milk, did make the observation that such feeding has no obvious beneficial effect in the diarrhoea of bacillary dysentery: 'In bacillary dysentery . . . starvation, even if prolonged, is likely to have but little effect in causing a cessation of the diarrhoea'.

Goldbloom[41] evidently had little fear of fat and advised that it be added to the commonly used skimmed milk in the treatment of diarrhoea. He wrote: 'The consistency of the stools depends on saponification of fat, and you cannot make soap without fat any more than you can make bricks without straw'.[18]

Chung[42] considered that relapses of diarrhoea resulted from the vagaries of the disease, and not from oral food and fluid load. Lanman[43] doubted that any particular milk had especial virtues in the management of gastro-enteritis, so did Karelitz;[44] and Mitchell[45] introduced milk feeding very early in the treatment of diarrhoea. Young and Rogers,[46] in writing 17 pages on 'Recent advances in gastro-enteritis' managed to avoid the mention of feeding at all!

Dean and Weinbren[47] found fat quite innocuous. They administered it to infants with kwashiorkor. I have also had the opportunity of giving 1½ oz. of cream daily to some six children with kwashiorkor and diarrhoea without ill effects. Chung and Viscorova[48] did elegant balance studies to indicate that food and fluid absorption in diarrhoea is directly proportional to the food and fluid intake, notwithstanding the increase in bowel contents, for this increase is of little consequence; what is important is the food and fluid absorbed and utilized.

Holt and MacIntosh[49] considered that in the treatment of mild diarrhoea there is no need to change the diet in any respect—though fluid intake should be increased. Holt,[49] and Moncrieff and Evans,[50] while repeating the usual details of milk modification and dilution in diarrhoea, were nevertheless not entirely convinced, for they did mention the work of Chung and Holt,[42] and suggested 'feeding a diarrhoea' as an alternative means of management. The 1954 edition of Nelson[33a] adopted the same neutral stand, but in the 1959 edition the relevant section was written by Cooke, who made it clear, in a few short sentences, that he disapproved of Chung's views.[33b]

A New Emphasis

Here the scene changes, however, and shifts from intestine to kidney. Milk dilution and modification has effects not only on the alimentary apparatus, but also on the excretory organs; and having established, as is now reasonably clear, that virtually all foodstuffs, including undiluted cow's milk, are well tolerated by the intestine, even when inflamed, it is now questioned whether these same foodstuffs are equally well tolerated by the kidney.

Among those who wrote on milk modification in gastro-enteritis, the more recent authors have been paying attention to renal function, so that over the past half-century there has been a gradual and subtle shift of emphasis from digestive intolerance to renal intolerance, and from the composition of milk to alterations in the composition of body fluids consequent on diarrhoea.

Light has also been thrown on the problem by advances in bacteriology and virology. Nowadays very little is heard in medical circles (though a great deal in lay and even nursing circles) concerning milk constituents as causative factors in diarrhoea. Instead, increasing attention is being paid to the rôle of micro-organisms in gastro-enteritis.

The rôle of salmonella, shigella and staphylococci as causes of gastro-enteritis has long been known, but it is only in the last 10 years, and especially in the last 5 years, that the rôle of enteropathogenic *Escherichia coli* (EPEC or EEC) has been appreciated. Widely divergent views on their aetiological significance have been expressed—probably mainly owing to the zeal with which the subject has been pursued. More than 20 strains[51] of EEC have been implicated in epidemics of diarrhoea, and presumably the more strains sought for, the more will be found. While many series of studies indicate that EEC may account for some 20-25% of cases of infantile gastro-enteritis,[52-55] one series in Russia implicated EEC in 50% of cases.[56]

In America in particular there are adequate reports[57-60] to indicate the remarkable ease with which newborn infants may be infected with EEC carried by their mothers, although these infants

do not necessarily show clinical diarrhoea, for reasons which are obscure. In one study of 360 parturient mothers, about 13% had EEC (11 strains tested) and in this group 40% of their infants had the selfsame EEC isolated from their stools within 4 days of birth.[58] In another study about 25% of infants, shortly after birth, exhibited the same *E. coli* serotypes as their mothers.[59] In Germany EEC were reported as being rife in mothers and nurses, and in breast milk given by bottle. Some 9% of mothers were carriers of EEC, and infant infection occurred shortly after birth, during birth, or perhaps even earlier.[60]

But if EEC are responsible for diarrhoea, their presence is not the only factor in the occurrence of diarrhoea. The food ingested also seems to be significant and may modify the course of the diarrhoea. *E. coli* and EEC grow differently in milk media containing added sugars. The addition of sucrose rather than glucose, maltose or lactose, favours the growth of EEC.[61] However, human milk produces an intestinal situation which is inhibitory to the growth of all types of EEC. It possesses a factor necessary for the establishment and maintenance of a high formic-acid level in the intestine, and a lower pH value—both factors being inimical to the growth of EEC.[62]

Just being considered as aetiological factors at present are the viruses, in particular the entero-cyto-pathogenic-human-orphan (ECHO) viruses.[63-66] It is conceivable, if not probable, that future investigations will show that these organisms play the principal rôle in the causation of infantile gastro-enteritis—at least in well-cared-for infants from good homes—but it seems unlikely that the type of milk diet will affect the viruses in the same way that it affects the growth of *E. coli* and EEC.

Milk alterations in diarrhoea are being relegated to a menial and secondary rôle, not only by virtue of the appreciation of the part played in diarrhoea by micro-organisms, but also by a better understanding of the profound biochemical alterations which may be induced by the diarrhoea, by vomiting, and by ill-considered treatment.

CLINICAL BIOCHEMISTRY

The states of electrolyte imbalance in gastro-enteritis have been considered elsewhere,[67] but it is necessary to review briefly the effects on the body fluids of milk modifications administered during the course of gastro-enteritis, and the rôle of the kidney in this scheme.

Finkelstein[3d] described infants wasting away from what he termed 'decomposition'. Twenty years later, the first glimmerings of the rôle of water in body metabolism were noted,[68] and it rapidly became clear that 'decomposition' was really 'dessication'. From this period (1920), fluid therapy has burgeoned to bewildering complexity, reflecting the increasing importance of clinical biochemistry in child care.

Finkelstein[3d] did tinker with the electrolytes of milk. His protein milk was low in sodium and potassium, for he hoped by this means to produce more solid-looking stools. However, deficiencies in electrolyte intake are not conducive to recovery from gastro-enteritis.

The addition of lactic acid to milk is theoretically bad. It may be responsible for causing some degree of metabolic acidosis,[69] and in severe diarrhoea there is no need to aggravate the metabolic acidosis already present.

Skimmed milk is used almost universally in infantile gastro-enteritis, the fat being considered harmful and indigestible. But Gomez observed that '. . . the fear of losing other nutrients, especially some minerals, by increasing the intake of foodstuffs in general, and of fat in particular, does not seem to be justified'. He continued '. . . it seems useful to insist that the long-established practice of either reducing or suppressing the intake of fat in steatorrhoea, particularly in malnourished children, seems to be unnecessary and might even be undesirable'.[70] Certainly I have never understood the use of skimmed milk in kwashiorkor, with or without diarrhoea, and cannot imagine that it is superior to whole milk for this purpose.

In recent years we have been hearing a great deal about osmolar loads in the presence of renal insufficiency. Despite a comparatively large extracellular-fluid compartment, the infant has a high rate of fluid turnover, perhaps two or three times that of the adult, and cannot tolerate fluid depletion. The young infant needs comparatively more fluid to excrete the same osmolar load. Older children and adults can concentrate urine highly and can excrete something like 1,400 m0sm. of solute per litre of urine. Premature infants, and newly born mature babies can only excrete some 600-700 m0sm. per litre, though by the second month of life they can manage 1,200 m0sm. per litre.[71,72] However, this is no great safety factor, for the fluid turnover is still rapid and the rate of metabolism high.

Fomon[73] has shown that infants up to 6 months being fed only on breast milk, excrete an average of about 100 m0sm. of solute per litre of urine, while those reared on reconstituted (unsweetened and undiluted) whole cow's milk excrete an average of about 500 m0sm. per litre of urine, the excess of solute being largely made up of phosphates and products of protein catabolism. Among prematures, or term infants in the first month of life, renal function, as judged by adult standards, is rather limited, for the kidney can only concentrate to about half the adult standard, so that such infants, fed on whole cow's milk, do not have much water available as a reserve in the event of environmental heat, fever, vomiting or diarrhoea. Breast-fed infants have 4 or 5 times the water available for solute excretion as compared with babies getting whole cow's milk. In the second month of life, however, kidney concentrating ability is markedly increased, reaching levels of about 1,200 m0sm. per litre, which is almost that of adult proportions (1,400 m0sm. per litre).

However, levels reached in health do not necessarily apply in sickness. In the acute phase of hypertonic dehydration, when there is urgent need for maximum solute excretion in minimum water volume, the infant kidney seems unable to concentrate to more than 400-700 m0sm. per litre, much below the theoretical possibility of 1,200 m0sm. per litre. The cause of this interference with normal expected water conservation in infants with diarrhoea is not understood, but might result from absolute potassium lack or the effects of the toxic products produced by the infecting organisms.[74]

Darrow[75] has made the important observation that solute excretion in infants should be seen in the light of calories metabolized, rather than in terms of patient weight or surface area, and has suggested that 100 calories metabolized should be adopted as a standard of reference.

Infants being treated on intravenous dextrose and electrolytes only (in the commonly used concentrations) produce somewhere around 20-25 m0sm. of solute per 100 calories metabolized, while infants on a conventional cow's milk and solid diet produce more like 35-40 m0sm. per 100 calories,[33b,75] the excess being mostly derived from phosphates and the products of protein catabolism. What is thus of importance, as far as solute load is concerned, is not the number of calories supplied, but the amount of protein per calorie supplied. In this context, protein takes on a new rôle, as a substance which may embarrass renal function at a time when its function may be already impaired consequent on the dehydration following diarrhoea, vomiting, hyperventilation, fever, and environmental heat.

Cooke[61] thus regards the use of skimmed milk in diarrhoea as unwise, for 'skimmed milk presents a high renal-solute load at a time when extra-renal losses of water are high'. The 6th edition (1954) of Nelson's *Pediatrics*[33a] advocates the use of skimmed milk, or skimmed lactic-acid milk in diarrhoea. The 7th edition (1959)[33b] now condemns this practice as being responsible for hypertonic dehydration, which 'occurs . . . particularly when the renal-solute load is high, as when boiled skimmed milk is fed to infants with diarrhoea'. Finberg considers the use of skimmed milk physiologically irrational for 'the load of electrolytes and protein (chief constituents of the solute load) *per calorie* is much greater in skimmed milk than in whole milk'.[34]

Reflecting this new view on osmolar relationships, recent advertisements for at least one type of milk preparation adopt a fresh and novel outlook. No longer is attention drawn to alterations in the carbohydrate, fat and protein moieties. Instead we find one advertiser praising his milk product thus: 'For fluid balance feed Bremil, because renal-solute load and water requirements are kept within normal limits by a physiological protein/electrolyte pattern comparable to that of breast milk. Particularly important during periods of febrile illness, diarrhoea, and in hot weather'.[76]

CONCLUSION

So the wheel of therapy has turned a full circle and more. In the past the presence of diarrhoea raised fears about the excessive content of protein, fat, carbohydrate and electrolytes in milk. The only milk constituent that was not blamed was the water. Today we know better, for even water may be dangerous, and water intoxication can be fatal. All that has been feared so much in the past is now, with grand illogic, freely given intravenously: sugar, protein, electrolytes; even fat is available for infusion into the veins.[77,78] Of these substances it is the water, the electrolytes, and especially the protein that may cause harm in the presence of complex biochemical disturbances.

It is clear, though, that fat is no longer to be regarded as causing 'intolerance'. On this charge fat must be exonerated. Let there be no equivocation about the matter: fat is good for diarrhoea. In general, fat tends to produce solid stools. It is common practice to restrict fat for as long as the stools are loose, and the stools commonly remain loose for as long as the fat is restricted. Fat is a vehicle for the intake of a number of vitamins. Catabolism of fat makes extra water available to the body—about 1·0 ml. of water per gram of fat metabolized, and in this respect it is twice as efficient as carbohydrate and protein. Fat supplies a high content of calories without producing a solute load on the kidneys. As it is, cow's milk is very inferior to breast milk as a source of essential unsaturated fatty acids[79,80] and tocopherol.[81,82] When partially skimmed cow's milk is given to infants from birth, their plasma level of vitamin E remains consistently low.[81] Half-cream milk is especially popular in England for the feeding of infants in the first few weeks. When it is considered that this practice deprives such infants of essential fatty acids and vitamin E, it is seen to be of dubious value. Moreover, it is done at a time when the kidney function is limited (by adult criteria), thus presenting a high solute load (of protein) per calorie ingested; and this form of feeding is especially to be deprecated in the presence of diarrhoea; it is quite unphysiological.[82]

In the treatment of diarrhoea the use of whole cow's milk (better still, breast milk) is indicated. There is no need to dilute it with water on the basis that somehow the milk is 'heavy' and hazardous, and aetiologically linked with the diarrhoea; and unless vomiting is present (with the danger of aspiration) there is certainly no need to stop milk altogether. Cow's milk is a valuable source of electrolyte in hypotonic concentration, viz. sodium about 25 mEq./l., chloride about 30 mEq./l., potassium about 40 mEq./l., and calcium, magnesium and phosphate are also present. Extra water must be administered in order to replenish body deficits and to enable the kidneys to function properly, but it is misleading especially to the parents, to insist on the dilution of the milk, for the milk is blameless. The water may be given separately. Though the result is the same, the concept differs.

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Medical Association of South Africa

FORTY-THIRD SOUTH AFRICAN MEDICAL CONGRESS

24 – 30 SEPTEMBER 1961, CAPE TOWN

SECOND CIRCULAR

Telephone: 3-3355
Telegrams: 'MEDKONGRES'
Cape Town

Medical Congress Office
P.O. Box 643
35 Wale Street
CAPE TOWN

GENERAL INFORMATION AND PROVISIONAL PROGRAMME

The Forty-third Medical Congress of the Medical Association of South Africa will be held in *Cape Town* on *24 - 30 September 1961* at the University of Cape Town, Rondebosch, Cape.

OFFICE BEARERS

There have, of necessity, been some changes. The President, Vice-President, and Honorary Vice-Presidents remain as published in the First Circular (p. 971 of the *Journal* of 12 November 1960). Prof. B. Bromilow-Downing has succeeded Dr. F. W. F. Purcell as Chairman of the Organizing Committee. Dr. Purcell has recovered from his illness and continues to serve on the Committee as an ordinary member. Other changes on the Organizing Committee and Sub-Committee, as well as Scientific Sections, will be published in the final Circular.

SUNDAY 24 SEPTEMBER

Registration. The Registration Office will be in the Students' Union at the University of Cape Town, and will be open on Sunday 24 September from 10 a.m. to 5 p.m. and on Monday 25 September from 8.30 a.m. Local members, as well as early arrivals from elsewhere, are requested to register as *early as possible*.

Registration fees are R10. Registrars will pay R6 and interns R2. Only members of the Medical Association of South Africa (M.A.S.A.) and affiliated associations may register. If resident in the Republic of South Africa, members of affiliated associations must also be members of the M.A.S.A.

Note. Members who have already paid their Registration Fees in full or in part (the R5 voluntary levy), have been credited with the amount paid, *but they still have to attend* for Registration.

The membership badge issued upon Registration *must be worn* at all meetings and functions, and by members when visiting clubs which have granted honorary membership facilities. The Congress brochure, diary, and other publications will also be issued at the time of Registration.

Tickets for the ball and/or banquet may be purchased separately upon registration.

Ladies' registration will be conducted concurrently, and the ladies will be required to indicate their choice of functions, excursions, sport, etc. at the time of registration.

Church service. The interdenominational church service will be, it is hoped, a most appropriate ceremony to initiate our Congress proceedings this year. The Diocesan College Chapel, Rondebosch, should prove to be an admirable setting. Roads will be signposted, and there is ample parking in the lovely college grounds. The choir will be in attendance. Academic dress may be worn—this is entirely optional. The service will commence at 6.45 p.m.

Public lecture. This will follow the Church service and will be delivered at 8.30 p.m. by Prof. J. F. Brock in the New Science Lecture Theatre at the University of Cape Town, Rondebosch. The President of Congress, Mr. J. A. Currie, will preside.

MONDAY 25 SEPTEMBER

Registration will continue from 8.30 a.m.

The opening of the Medical and Surgical Products Exhibitions, the Scientific Exhibition, and the Hobbies Exhibition will take place in the morning.

First television programme. As mentioned in the First Circular, closed-circuit colour television should prove to be one of the highlights of Congress, and the first programme to be presented, commencing at 12.30 p.m., should be able to demonstrate dramatically and most appropriately the value of this medium of instruction.

To the majority of the members of Congress, an open-heart operation is still a 'must' amongst things to see. Here, on a 9 × 12-ft. screen, you will virtually be looking over the shoulder of Dr. C. N. Barnard, who will be performing closure repair of a ventricular septum defect, using the heart-lung machine in conjunction with profound hypothermia. He will, however, be in an operating theatre in a hospital several miles away, and you will be sitting in the large auditorium at the University of Cape Town.

Group meetings and first scientific meeting. From 2 - 5.30 p.m. on Monday afternoon, there will be Sectional Meetings and combined meetings, details of which have not been completed. There will also be time for meetings of some of the National Groups.

Second television programme. From 4 - 5.30 p.m., the Paediatric Departments of the Universities of Cape Town and Stellenbosch will jointly present a televised paediatric programme under the direction of Dr. John Hansen. Before the cameras at the Red Cross War Memorial Children's Hospital, his team will demonstrate, and you, sitting in the auditorium, will see: Problems of nutrition in the pre-school child, management of prematurity in hospital and at home, practical procedures in paediatrics, respiratory emergencies, treatment of burns, etc.

The opening ceremony of congress and adjourned annual general meeting will be held in the City Hall on Monday evening. His Worship the Mayor will welcome members of Congress to the Mother City, and the President of Congress will deliver his presidential address. The opening ceremony will be followed by a Mayoral reception in the Drill Hall. A buffet supper will be served. *Academic dress:* It is hoped that members will wear academic dress at the opening ceremony though doing so will be optional.

TUESDAY 26 — FRIDAY 29 SEPTEMBER

A comprehensive programme of scientific meetings, further television presentations, and exhibitions, as well as sport and entertainment have been arranged. Details will be published in the final circular. The following will give prospective Congress-goers an idea of what they will be able to see, hear, and do during Congress week:

Plenary sessions and combined meetings. Plenary Sessions will be two in number and will be entitled *Care of the aged* and *Diabetes*. The mornings of Tuesday and Wednesday, respectively, will be devoted to these meetings. During the afternoons of these two days, these themes will be continued as combined sectional meetings, open to sections with particular interest in these subjects, while other sections will be having their own meetings concurrently.

On Thursday morning, *The diagnosis and treatment of occlusive vascular disease*; on Friday morning, *Respiratory failure*, and on Friday afternoon, *Antibiotics, chemotherapy and cross-infection*, should prove to be combined meetings of exceptional interest.

Sectional meetings will be held concurrently with these by those

sections not taking part in combined meetings. In addition to the above, two symposia on *Rheumatic heart disease* and *Pulmonary and pleural neoplasia* are being arranged.

Group meetings: These will be fitted in to suit Groups.

Television programmes: In addition to the two given on Monday, 6 further programmes are being presented.

Tuesday 4 - 5.30 p.m.	A radical mastectomy, by Prof. J. H. Louw.
Wednesday 4 - 5.30 p.m.	Gastrectomy, by Dr. B. J. van R. Dreyer.
Thursday 2 - 3.30 p.m.	Vaginal hysterectomy, by Prof. J. T. Louw.
Thursday 4 - 5.30 p.m.	(*a*) Total abdominal hysterectomy, by Prof. J. N. de Villiers. (*b*) Lower segment caesarean section, with demonstration of resuscitation of infant, by Dr. P. F. M. du Toit and Dr. R. F. Maggs (paediatrician).
Friday 12.30 - 2.00 p.m.	Cholecystectomy, Mr. R. L. Forsyth.
Friday 4 - 5.30 p.m.	(*a*) Demonstration of ambulant treatment of varicose ulceration; and (*b*) Radical varicose vein operation, by Dr. J. J. W. van Zijl.

The operations will all be performed and televised at the Red Cross War Memorial Children's Hospital, and screened at the large auditorium at the University of Cape Town.

EXHIBITIONS

1. The exhibition of Medical and Surgical Products has become an integral part of Medical Congress. The Jameson Hall and adjacent Lower Sports Hall will house the exhibits. All the exhibitors have been involved in great expense, and it is our duty to make it worth their while and to show our appreciation by visiting these exhibits.

2. The Scientific exhibits are being held in the large new Zoology laboratory and, as at the 1949 Congress, are in the able hands of Dr. H. O. Hofmeyr, who has had wide experience in arranging exhibits of this nature. Members wishing to exhibit may contact him at the Congress Office. Apart from static exhibits, the exhibition will include films.

3. The Hobbies exhibition will be held in the Senate Room in the Arts Block. As in 1949, Mr. T. Schrire is the convener. Members who have already signified their intention to exhibit, and those who still propose to do so, are requested please to forward a short description of their exhibit *immediately*.

ACCOMMODATION

Accommodation will be handled exclusively by the South African Railways Travel and Publicity Bureau, who have been appointed the official accommodation agents for Congress. For information in connection with bookings, members are asked to contact their local agencies. Applications will be attended to promptly and forwarded to the controlling office in Cape Town. In the event of difficulty, kindly contact Dr. J. C. Coetzee, the Convener of the Accommodation Sub-Committee, at the Congress Office, 43rd South African Medical Congress, P.O. Box 643, Cape Town.

Transport in Cape Town. This will be arranged where required for visitors. The Convener is Mrs. J. D. Joubert.

Banking and postal facilities will be available.

Catering. Tea and coffee with snacks will be obtainable free in the Students' Union daily at 11 a.m. and 3.30 p.m. Lunch will be served daily at a charge of 50c.

INFORMATION BUREAU

This service is being run for us, as at previous Congresses, by Caltex (Africa) Ltd., who will issue badges, car stickers, pocket diaries, etc. and will deal with all inquiries and requirements of members. The Bureau will be housed in the 'Varsity' Room at the South-east corner of the Students' Union, facing the Jameson Hall steps.

Signposting of roads will also be done by Caltex.

ENTERTAINMENTS

The Ball will be held in the City Hall on the night of Wednesday 27 September. Tickets will be sold for R6 double.

The Banquet takes place in the Weizmann Hall on Thursday 28 September, and is open to **wives of members as well.** Prices are R9 per double ticket and R5 single, inclusive of wines and cigarettes.

On the night of Tuesday 26 September, the very popular Caltex Show and Kaskenades will be presented.

The Woodstock Town Hall has been booked for Friday night for the presentation of a Khalifa.

LADIES' ENTERTAINMENTS

An attractive programme has been arranged which includes excursions all over the Western Province, a mannequin parade, etc.

Monday afternoon: Visit to Peninsula factories of special interest to women.

Monday evening: Opening ceremony and Mayoral supper.

Tuesday afternoon: Mannequin parade and champagne cocktails at the Clifton Hotel.

Tuesday evening: Caltex Show and Kaskenades.

Wednesday morning: Sightseeing in and around Cape Town, including cableway trips to the top of Table Mountain, weather permitting.

Wednesday afternoon: Free.

Thursday morning: Free.

Thursday afternoon: Sports afternoon and excursions to the KWV at Paarl and Protea Heights at Stellenbosch, with refreshments at Stellenvale Winery at Devon Valley.

Thursday evening: Congress banquet.

Friday morning: Excursions to Vergelegen, Somerset West, or Constantia and Kirstenbosch.

Friday afternoon: Free.

Friday evening: Khalifa.

SPORTS DAY

This will be on the afternoon of Thursday 28 September, and the usual men's and ladies' golf and bowls tournaments, etc. will take place. Facilities for the following sports at any time during Congress week are available for those who wish to do so: golf, bowls, tennis, fishing, swimming, yachting, etc.

Fishing. It is not the tunny season, but those members who would like to sample rock angling or deep-sea fishing, can do so in small parties and under the guidance of experts, provided the weather is favourable.

Tennis. Courts are being made available at Kelvin Grove, King David Country Club, Keurboom Sports Club, and the University of Cape Town, as well as privately.

Swimming. Quite a few members have expressed the wish to indulge in aquatic sports, and the University Baths will be made available.

Squash. Facilities for playing squash will be made available at the University of Cape Town courts

Racing. The following meetings are being held at Congress time: 23 September at 1.30 p.m.—Kenilworth; 27 September at 1.30 p.m.—Durbanville; and 30 September at 1.30 p.m. at Kenilworth. Honorary membership facilities have been extended to members of Congress for the purpose of attending these race meetings.

CLUBS

The following Clubs have extended facilities of honorary membership to members of Congress:

Mowbray Golf Club, Westlake Golf Club, Royal Cape Golf Club, Rondebosch Golf Club, Metropolitan Golf Club, Milnerton Golf Club, King David Country Club, Keurboom Sports Club, Clovelly Country Club, Royal Cape Yacht Club, Zeekoevlei Yacht Club, Cape Aero Club, South African Turf Club (Kenilworth), Durbanville Turf Club, City Club, Alexandra Club (Ladies), Klub Here Sewentien, Civil Service Club.

INTENTION FORM

An Intention Form is printed on page xxv of this issue of the *Journal*. Please complete and forward it to the undersigned at the above address.

JACQUES ROUX, M.B., CH.B., M.R.C.O.G.
Hon. Organizing Secretary

VIRUS DISEASES IN GENERAL PRACTICE*

D. H. GIRDWOOD, *Bedford, Cape*

I have chosen this subject because, when I qualified in 1940, viruses occupied a small space at the end of books on bacteriology. In the last 20 years, with the antibiotic control of bacterial infections, virus diseases have taken a much more prominent place in clinical practice. A great deal of research has been and is being done on viruses, much of it in this country, and yet, apart from the specific infectious fevers, very little clarity has emerged about the clinical syndromes associated with infection by the different viruses. Different viruses seem capable of producing similar febrile illnesses, and the same virus can produce totally different clinical manifestations among patients—even in the same family.

Dr. Girdwood

A great deal of the work, which aimed at determining the relation between differing patterns of clinical illness and the virus and bacterial agents associated with them, has been disappointing from our point of view in that a clear-cut picture has not emerged. For example, in respiratory diseases in children, the potential bacterial pathogens isolated have been of much the same order as have been obtained in surveys of healthy children. In only a relatively small percentage of cases were viruses isolated, and it was not possible to predict the virological findings from the clinical picture in most of them. What has emerged, however, is the fact that in endemic respiratory illness, including tonsillitis and pharyngitis, about 10% are streptococcal throat infections, 5% other bacterial infections, and the rest, i.e. about 85%, viral infections or infections presumed to be caused by viruses. It has been estimated that between 20 and 30% of all visits by general practitioners to the homes of their patients are to attend to infections caused by viruses. Most virus diseases are benign and self-limiting; some, like poliomyelitis, are crippling and others, like influenza, are sometimes fatal. One disease—rabies—is almost invariably fatal. The fact is that virus diseases are at present one of the major hazards of life.

May I briefly summarize some of the facts in the virus research of the last 20 years that might be of interest to general practitioners. We know that whereas we regarded viruses in the past as miniature bacteria, it is more appropriate to regard them as relatively complex chemical structures comprising a protein shell surrounding a nucleic acid. Multiplication of viruses is only possible within the cells of the host. In simple terms this is achieved by the virus' affinity for the genetic apparatus of the invaded cell, and the utilization of the cell metabolism for its own needs. In some way the invaded cell is stimulated to produce virus protein and virus nucleic acid. This might destroy the cell in the process or the virus might remain dormant for years and only produce symptoms when disturbed by some stimulus. This is exemplified by the virus of herpes simplex, which may invade the body during infancy and remain dormant until an attack of pneumococcal pneumonia or cerebro-spinal fever precipitates the eruption of a crop of vesicles on the lips, caused by this virus.

Laboratory Findings

Twenty years ago 36 antigenetically distinct human viruses were known, and many of these could not be grown in the laboratory. Now more than 150 distinct human viruses are known, only 5 of which have not been grown in the laboratory. Virologists have made major advances in the technique of tissue culture. Living tissues of various kinds, with nutrient media and antibiotics to eliminate bacterial contamination, can be grown in test tubes, and viruses made to grow in these cells. Certain viruses can only grow in one type of tissue culture. Others produce recognizable change in the cells, for example poliovirus produces cell degeneration within a few hours—so-called cytopathogenic change—which can be recognized readily under a microscope. This change can be prevented by adding polio-antibody. The laboratory can therefore help us in the diagnosis of viral infections by the direct method of isolating the causal virus from pathological specimens, and secondly by the demonstration of antibodies in the serum in convalescence. To be significant, a four-fold increase occurring during convalescence would need to be demonstrated. In practice the laboratory cannot help us here, since we are 600 miles from the nearest centre able to undertake the work. Even if available it would help us only in outbreaks of obscure virus disease, and then retrospectively. It behoves us therefore to try to recognize viral infections on clinical grounds alone, together with simple side-room methods.

Clinical Picture

Are there any positive signs of a viral infection as distinct from the negative one of failure to respond to antibiotics? Undoubtedly there are. Before considering them, it might be worth while looking at the poliovirus, about which we probably know most. In the first stage, after ingestion or inhalation of the virus, it proliferates in the lymphoid tissue of the pharynx and tonsils, and also in the intestine, especially in Peyer's patches. In the second stage, the virus passes to the regional lymph nodes, where further multiplication occurs. In the third stage, virus enters the blood stream, producing a viraemia lasting 4 or 5 days. This stage is usually accompanied by fever and a non-specific illness. Finally, the virus enters the target organ—the central nervous system.

This biphasic quality of non-specific minor illness with general constitutional symptoms, headache, general body pains, pharyngitis, nausea and vomiting, followed later by symptoms of disordered function of an organ, appears to be a feature of many virus infections, e.g. infective hepatitis, measles to some extent, and aseptic meningitis, and it is worth bearing in mind as a positive feature of a viral infection.

Other positive clinical features of a viral infection which are often present are: leucopenia; bradycardia relative to the temperature; a tendency for viral infections to occur in epidemics; a tendency for different clinical manifestations to occur in the same epidemic; and a usually prolonged convalescence.

Types of Viruses

May I mention a few of the viruses discovered in the last 20 years that are of interest and importance to us. Firstly, there are the family of enteroviruses, so-called because they multiply freely in the gastro-intestinal tract. This family comprises the poliovirus, the Coxsackie Group A and B, and the ECHO viruses. There are many different types in this group. Of the 150 virus types mentioned earlier, 52 are of this family. The Coxsackie A virus produces the characteristic syndrome of herpangina, which I will mention later, and the Coxsackie B, Bornholm's disease, which I think you will agree is a very common illness in practice. It has also been found to produce myocarditis in the newborn, hence its potential danger in an epidemic in a family. The ECHO virus produces no very characteristic syndrome—it has been found in various nondescript febrile illnesses in children occurring during the summer months, often associated with a rash. It also causes minor respiratory illnesses, and diarrhoeal illnesses. More important is the fact that these 3 enteroviruses can be the cause of aseptic meningitis, a benign form of meningitis occurring not too infrequently in practice. Most of the cases are probably diagnosed as abortive poliomyelitis. The characteristic features of the cerebrospinal fluid are the presence of cells, a sterile culture, and a normal glucose content. In any of the pyogenic meningitides, in contrast, the glucose would be diminished.

The next group to consider are the adenoviruses, which probably cause a fair amount of the endemic non-specific respiratory infections, as well as pneumonitis. One type causes epidemic keratoconjunctivitis, which is fairly common among the Bantu.

Finally, the group of myxoviruses, which include the influenza viruses, the mumps virus and various other viruses which seem able to cause upper respiratory and croupous infections. An interesting feature about influenza A virus is that whereas most other viruses

* Presidential Address, Cape Eastern Branch, delivered at Annual Meeting, 6 May 1961.

are, as far as we know, antigenically stable, and therefore produce immunity, the influenza virus is antigenically unstable, and a constantly changing antigenic quality from one epidemic to another lays us open to 'flu' again and again when a new strain emerges.

Throat Infections

May I now discuss a symptom which is probably one of the commonest in general practice, namely sore throat. Leaving out obscure causes like agranulocytosis, and not mentioning diphtheria for which all general practitioners in this country are constantly on the watch, the problem largely resolves itself into the decision whether the condition is a streptococcal throat or a non-streptococcal viral infection. On purely clinical grounds it is often difficult to differentiate between them, but there are certain pointers which help.

The streptococcal throat will usually show a yellow-white exudate on the tonsils or pharynx, redness of the mucous membranes, adenopathy, a leucocytosis of more than 13,000, an abrupt onset, and subsidence in 24 hours with penicillin.

In the non-streptococcal exudative tonsillitis or pharyngitis, caused by adenovirus infection in some cases, and by an unknown virus in others, the throat itself looks much the same, except that a granular appearance of the posterior pharyngeal wall from hyperplastic lymph follicles seems to be a feature of this condition. Glandular enlargement is more likely in the posterior cervical region, the white cell count is less than 13,000, and there is often a cough, hoarseness, and sometimes conjunctivitis associated with the symptom of sore throat.

Infectious mononucleosis often presents with a sore throat, which can look very unpleasant with a necrotic-looking exudate. The adenopathy is usually prominent and in other sites as well as the neck.

In herpangina due to Coxsackie A virus, there are, characteristically, small vesicles on the tonsillar pillars or soft palate which later become ulcers with a surrounding areola, a normal white cell count and fairly prominent constitutional symptoms, such as headache, muscle pain, etc. The fever reaches its peak in 24 hours and then gradually returns to normal by the third day.

In contrast to this is the condition of herpetic gingivo-stomatitis due to the herpes simplex virus, in which the vesicles becoming ulcers are situated on the tongue and the buccal and sublingual mucosa. They have no surrounding areola and last for 10 - 16 days. Here also there is a normal white cell count. Recently, fatal cases of this infection in malnourished non-European children, following a general systemic dissemination of the virus, have been described in the *South African Medical Journal*.

Somewhat similar is Vincent's infection, but here there is usually a grey necrotic exudate on the tonsils as well as gingivitis, no adenopathy as a rule, and initially a normal white cell count.

I have observed the last 50 patients I have seen with pharyngitis and fever more critically. I made clinical notes, did leucocyte counts, and took throat swabs from most of them. A few interesting points have emerged. All those with swabs positive for haemolytic streptococci had white cell counts of 13,000 or more (on an average 16,000). Response to penicillin within 24 hours was the rule. Other symptoms and signs were not uniform—these included rigors, suddenness of onset and the appearance of the throat. Many patients with yellow, spotty exudate on inflamed tonsils had negative cultures and low white cell counts; these were presumably viral in origin. Granularity of the posterior pharyngeal wall from enlarged lymphoid follicles was a feature of non-streptococcal infection.

Two of the patients had diphtheria, and both of these had white cell counts of more than 20,000. In the patient with the highest count of all, 25,000, the organism grown was the pneumococcus.

Only 9 of the 50 patients had positive haemolytic streptococcal cultures, but another 10 were almost certainly streptococcal in nature. This low rate of positive swabs may be the result of the far from ideal arrangement, from the bacteriological viewpoint, of sending swabs to East London, with resultant delay. The incidence, then, of streptococci in this small series was 38%. Just on 50% of the patients had low or normal white cell counts, and many of them had positive features of a viral infection.

Abuse of Antibiotics

You might well ask what is the point in making these distinctions. The treatment, after all, is the same—antibiotics. It is here that I wish to make the main point of this address. No known antibiotic or chemotherapeutic agent has any action whatever on the viruses. Surely it is not only wasteful, but also positively harmful, to treat known virus infections with antibiotics, particularly the broad-spectrum ones.

When treating an exudative tonsillitis or pharyngitis, it would seem reasonable to give penicillin, even though there is only a 25 - 50% chance of the condition being a streptococcal infection. Take a swab at the same time and, if streptococci are present, treat for 10 days in order to eliminate the organism and prevent rheumatic fever. The tetracyclines and sulphonamides, because they are bacteriostatic and not bactericidal, are not indicated. In the other non-specific respiratory infections and febrile illnesses, surely it would be wiser to withhold antibiotics and to observe these patients intelligently, treating complications that might arise, particularly in the very young.

With regard to the prophylactic use of antibiotics to prevent secondary bacterial infection in the course of these virus illnesses, controlled studies, using penicillin as the antibiotic, have shown that the illness in the treated patient lasts longer than in those left untreated. Similarly, a survey of the prophylactic use of antibiotics in measles, undertaken in England by the College of General Practitioners, has shown an increased morbidity in the treated patients, compared with controls. I have come to a similar conclusion in giving sulphonamides to Bantu children with measles.

Those of us, like myself, who began practice in the antibiotic era, are apt to forget that infections can, and do, subside without antibiotics. It is salutary to read, on occasion, medical text books written about 50 years ago. For example, Thompson, in the section on croupous pneumonia in his book *Clinical Examination and Treatment of Sick Children*, says: 'The prognosis is very favourable considering how ill the child generally appears to be . . . The disease is self-limited and has a strong tendency towards recovery'.

The antibiotics were a life-saving discovery and surely should be used to save life rather than to treat fever simply as expensive antipyretics. How often does one's phone not ring and an anxious mother ask: 'Doctor, my child has a temperature of 101°. I have 'achromycin' in the house. May I give it?' It is not easy to resist the pressure brought to bear by an anxious parent. Why not be on the safe side, one says, and give a little achromycin or whatever antibiotic she happens to have? It won't do any harm. But surely we are actually doing a disservice. We may mask a urinary-tract infection, for example, if we give antibiotics before local symptoms have declared themselves, and thus treat it only partially.

In bigger centres even cold elective surgery is hazardous in the face of the menace of the resistant staphylococcus. Which one of us hasn't had a terrifying moment with drug-sensitivity reactions? These problems will increase by multiple progression until we can control the abuse of these drugs. Most of us do not treat our own children unnecessarily with antibiotics. In most cases, by the time we get round to looking at them when they are sick, their illness is on the wane, and they are happily spared treatment.

The Future

Finally, what of the future? With the present tempo of research on viruses, the next 20 years should have fascinating possibilities.

That viruses and cancer may be related is an old observation. The theory that in most human beings some sort of cancer virus becomes established and remains entirely latent, except in a few unfortunate individuals in whom additional factors result in a disturbance of the balance, is a hypothesis only and an old one. However, recent research tends to substantiate rather than detract from this theory, and clearly the development of chemotherapy for virus diseases and cancer is along the same path of research, owing to the similarity of the virus-infected cell and the cancer cell. The key to this problem might be found by the biologist and organic chemist in their work on genes, viruses, cancer and nucleic acids.

In our own humble sphere, which is none the less important, there is need for observation and more accurate definition of the very many febrile syndromes occurring in practice, and the noting and publication of these so that others may recognize them. This can only come about by some direction from above, for example by those undertaking virus research, or by an epidemic-observation unit of the College of General Practitioners. This is a very necessary step to bring some order into the chaos of virus diseases.

UNIVERSITEITSNUUS : UNIVERSITY NEWS

UNIVERSITY OF THE WITWATERSRAND : EXAMINATION RESULTS

The following candidates have completed all the requirements of the Sixth Professional Examination for the degree of M.B., B.Ch.:

Blekie, E. V. Kirsch, P. E. Perlman, M. M. Chothia, A. A. Dalamba, K. Eidelman, J. L. Ho Shon, J. Kriek, J. C. le Roux, J. S. Morris, M. S. Price, A. R. Robertson, C. Shaff, M. I.

IN DIE VERBYGAAN : PASSING EVENTS

Dr. Samuel Hoffman, of Johannesburg, has been elected a Fellow of the Faculty of Anaesthetists of the Royal College of Surgeons in Ireland.

* * *

British Medical Association: Competition for Medical Films. In order to encourage the production of medical films, the Council of the British Medical Association is prepared to award medals and certificates for 16mm. medical films to be submitted in open competition. Awards will be considered in two categories—for commercial and for non-commercial film producers. The choice of subject is left to the competitor.

Entry forms and further particulars may be obtained from the Secretary, British Medical Association, B.M.A. House, Tavistock Square, London, W.C. 1. The closing date for entries is 30 September 1961.

* * *

Mr. B. de Saxe, surgeon, of Johannesburg, has returned from a visit to the United States where he attended surgical clinics in New York, Boston, New Haven, and Washington.

* * *

University of Natal, Sub-Department of Paediatrics. The next meeting will be held on Tuesday 11 July in the Medical School, Durban, at 8 p.m. Prof. D. S. Chapman will speak on 'The surgical emergencies in the newborn'.

* * *

Dr. S. Lopis, specialist physician, of Johannesburg, has left on a visit overseas to attend Endocrine and Diabetic Units, and the World Diabetic Congress in Geneva.

* * *

Nutrition Society. A meeting of the British Nutrition Society will be held at the National Institute for Research in Dairying, Shinfield, Reading, on Friday 13 October 1961. The meeting will take the form of a Symposium on 'Agricultural problems in underdeveloped countries'. The Chairman of the meeting will be Prof. R. G. Baskett. Those practitioners who may be in Britain during October and are interested in attending this meeting, may obtain further information from D. F. Hollingsworth, the Hon. Programme Secretary, c/o Ministry of Agriculture, Fisheries and Food, Great Westminster House, Horseferry Road, London, S.W. 1.

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 17 July at 5.10 p.m. in the Institute Lecture Theatre. Drs. H. E. Reef and C. Isaacson will speak on 'Atherosclerosis in the Bantu'.

* * *

Margaret Orford Memorial Lecture 1961: The Council of the College of Physicians, Surgeons and Gynaecologists of South Africa announces that the Margaret Orford Memorial Lecture for 1961 will be given by Prof. O. S. Heyns, Professor of Obstetrics and Gynaecology, University of the Witwatersrand. The title of the lecture is: 'A device to improve foetal oxygenation' and the dates and venues are as follows:

Bloemfontein: Tuesday 18 July 1961, 8 p.m. Lecture Room, National Hospital.

East London: Thursday 20 July 1961, 8 p.m. Recreation Hall, Nurses' Home, Frere Hospital.

Port Elizabeth: Friday 21 July 1961, 8.15 p.m. Nurses' Lecture Theatre, Provincial Hospital.

Cape Town: Wednesday 26 July 1961, 8.15 p.m. Physiology Lecture Theatre, Medical School, University of Cape Town.

Members of the College and all other interested medical practitioners are invited to attend these lectures.

Margaret Orford Gedenklesing 1961: Die Raad van die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika, kondig aan dat die Margaret Orford Gedenklesing vir 1961 deur prof. O. S. Heyns, Professor van Obstetrie en Ginekologie, Universiteit van die Witwatersrand, gelewer sal word. Die titel van die lesing is: ‚A device to improve foetal oxygenation', en die datums en vergaderplekke is soos volg:

Bloemfontein: Dinsdag 18 Julie 1961, 8 nm. Lesingsaal, Nasionale-hospitaal.

Oos-Londen: Donderdag 20 Julie 1961, 8 nm. Ontspanningsaal, Verpleegstershuis, Frere-hospitaal.

Port-Elizabeth: Vrydag 21 Julie 1961, 8.15 nm. Verpleegsters-Lesingsaal, Provinsiale-hospitaal.

Kaapstad: Woensdag 26 Julie 1961, 8.15 nm. Fisiologiese-Lesingsaal, Mediese Skool, Universiteit van Kaapstad.

Lede van die Kollege en mediese praktisyns wat belangstel word uitgenooi om die lesings by te woon.

BOEKE ONTVANG : BOOKS RECEIVED

The Use and Training of Auxiliary Personnel in Medicine, Nursing, Midwifery and Sanitation. Ninth Report of the Expert Committee on Professional and Technical Education of Medical and Auxiliary Personnel. World Health Organization: Technical Report Series, No. 212. Pp. 26. 17½c. Pretoria: Van Schaik's Bookstore. 1961.

Clinical Obstetrics and Gynecology. Vol. 3, No. 4. Fetal Physiology and Distress: edited by Thaddeus L. Montgomery, M.D. and Endocrinology: edited by Robert B. Greenblatt, M.D. Pp. 327. Illustrated. The series is published quarterly and is sold by subscription only for $18.00 a year. New York: Paul B. Hoeber. 1960.

BRIEWERUBRIEK : CORRESPONDENCE

CRITICISM AND DEMOCRACY

To the Editor: Dr. Marquard de Villiers' letter[1] in the *Journal* of 17 June evokes disturbing thoughts. For some time I have been concerned at the fairly regular appearance of letters in the *Journal* which seem to have as their purpose the furtherance of a smearing campaign against a person or persons who generally remain anonymous. However, it is never particularly difficult to identify the individuals concerned. This is a matter which disturbs me not only from the angle of personal taste of the writer concerned, but also that of editorial policy.* Very often these attacks are made against individuals who include amongst their number members who have served the Association for decades and occupied the highest offices of the Association. The fact that these critics of the Federal Council, or of some of its policies, or even of its august Executive Committee, constitute a minority within the Association, does not necessarily brand them as agitators, nor does it deserve the contemptible suggestion that personal motives might be involved.

A study of history in general, and of the Association in particular, shows repeatedly that the views of an unpopular minority at one stage in evolution have become in the course of time the accepted and popular view of the majority. There can be no better example

[*The correspondence Columns of the *Journal* serve the purpose of an open forum, and the views expressed in these columns do not necessarily reflect editorial policy–Editor.]

of this than the relatively recent history of the dispute over free hospitalization in the Transvaal. During the years 1946 - 48 the small band of critics of official Association policy were the targets of every form of attack and vilification. But, as events unfolded, so the Association came to realize that, by adopting the views of this minority, its own interests and desires were fulfilled. Dr. de Villiers would do well to study the history of the matters upon which he has permitted himself to express himself. Perhaps he might also reflect that his observations might ill become an individual who has attended but one Federal Council meeting. Whilst his narcissistic infatuation with the Central Committee for Contract Practice (of which he is a member), and in whose omniscience and infallibility he has such absolute confidence, is understandable, Dr. de Villiers should realize—as all new boys on the Federal Council should also realize—that the Association is still a democratic body; and that criticism, however unpalatable it may be, is the very essence of democracy.

Jonathan Gluckman

Jeppe Street
Johannesburg
24 June 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 507.

MEDICAL AID FEES

To the Editor: I am a general practitioner in active general practice and hold a part-time benefit society appointment. I agree wholeheartedly with Dr. Marquard de Villiers. The majority of general practitioners are deeply indebted to the Central Committee for Contract Practice.

The present medical aid fees are more than 4 times what I get for domiciliary and consulting room visits in my contract work. The new medical aid fees are almost as high as my private fees with this difference that, whereas in private practice the patients cannot afford the luxury of a second or third visit, in medical aid work one can pay these visits if they are really essential. Personally I would be pleased if my entire practice was medical aid practice. General practitioners in Houghton may disagree with this point of view, but I do not conduct a social bedside-manner practice, and my practice is amongst the middle-class population.

I also wish to assure Dr. de Villiers that not all of us are so gullible as to believe all the 'clap trap' that is being dished out by a certain small vociferous minority, most of whom are not in active general practice. Dr. de Villiers must credit us, the practising general practitioners, with a certain amount of common-sense and judgement, even if we do not take part in this continuous nerve-racking tub thumping. We also realize the dangers inherent in a more than likely State Medical Service when the present set-up will have been considered an Utopia.

A referendum would clearly indicate to the profession that the large bulk of active general practitioners—and I may add specialists, agree with the desirability of an extension of the medical aid scheme.

If Federal Council could only succeed in substituting the medical aid idea for all present contract practitioners, such as Railways, Mines, Municipalities, etc. the profession would truly have reason to be thankful to its elected representatives.

Medico

Johannesburg
17 June 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 507.

THE MEDICAL SERVICES PLAN

To the Editor: You have published a letter to Dr. Turton, dated 11 March, signed by Dr. M. Shapiro as Chairman of the Board of 'The Plan'.[1]

This letter appeared on the agenda of the meeting of the Executive Committee of Federal Council which was held on 21 April. It can be seen that it insinuates sinister motives to the fact that my letter in the *Journal* on 4 March was published so shortly before the Extraordinary General Meeting, also that my letter contained much erroneous and misleading matter in order to discredit the Plan.

I resented those insinuations and, since I could not attend the meeting of the Executive, I wrote a reply, to be placed before the Committee. When Dr. Shapiro circularized that statement by the Board to the whole of Federal Council, I sent my reply to all members, as well as to members of the Board of the Plan.

As I do not want to take up too much of your space and since I consider that these matters are more properly discussed in Federal Council, I crave your indulgence in allowing me to quote a few paragraphs from that reply. You, Sir, can of course bear me out that the late date of publication of my letter of 4 March, was none of my doing.

> I quote: 'The object of my letter (4 March) was not to discredit the Plan. That is borne out by its content, where I stated merely that the Plan had come too late in the history of medical insurance in this country. What I did want to do, was to prevent the Extraordinary General Meeting from stampeding the Association into itself adopting the Plan. Surely an Extraordinary General Meeting was not the place in which to discuss serious matters of policy of the Association. . . . Does not the Federal Council, after due deliberation, lay down policy . . . and not a few members, who come to an Extraordinary General Meeting with pockets full of proxies?'

The matter, which seems to worry Dr. Shapiro and the Board most, seems to be the paragraph wherein I mention 'the close on £3,000, already spent, to be considered a debt of honour to be repaid when the Plan was on its legs.'

I am not concerned with the *amount* of money involved, and that may have been unwittingly exaggerated. If that is the case, then I apologize. I do, however, maintain that Dr. Shapiro on more than one occasion in Federal Council, and in one capacity or another, gave his personal assurance that the Association would, at some time or other, be re-imbursed. It is not alleged that he is insincere, but he is often carried away by his own enthusiasm and optimism.

Let me quote what he said in Federal Council, October 1958, as Chairman of the reconstituted Sub-committee on the Economics of Medical Practice. This appears in a memorandum drawn up by Dr. Tonkin in June 1959 on 'The Development of the Medical Services Plan':

> 'Dr. Shapiro said: From the outset—and Federal Council actually resolved to advance £1,000 and expended a considerable amount of money in Mr. John's tour overseas, and that really is what influenced Federal Council in not proceeding with the matter at the time—I gave an undertaking that one of the first duties of the Fund, as soon as it can afford it, is that it should refund to the Association all the monies which it has expended to date. I still feel it is a matter of honour that this Fund should, in due course, return to the Association all the monies which it has expended directly or indirectly in the sponsorship of the scheme.'

At that time he was speaking to a motion to rescind the previously promised sponsorship of the scheme by the Association. Further assurances were obtained from him that 'sponsorship' meant 'blessing' and nothing more. In that favourable atmosphere then, most of the opposition to sponsorship was withdrawn. My vote, however, was recorded against sponsorship.

I held the opinion that sponsorship, however defined, would entail some moral responsibility on the part of the Association to see that the Plan did not fail and would, at all times, be able to fulfil its obligations to the public. A great financial burden might have to be shouldered, and I am still firmly of the opinion that it is not the business of a Medical Association itself to underwrite medical insurance for the public.

Furthermore, in my statement to the Executive, and now to the whole Council, I state:

> 'The Plan is emphatically Dr. Shapiro's baby . . . and he can hardly disclaim moral responsibility. . . . Nobody is such a fool as to think that he could, or did, legally bind the Plan, as it exists today, to repay any of that 'close on £3,000'.
>
> The Plan was a brave attempt, but many of us were of opinion that, in the existing state of development of medical aid insurance in South Africa in collaboration with our Association, it was inopportune . . . I do not begrudge the Plan its initial success in a circumscribed area, but I do think that we should hold it to its promised bargain of not claiming from the Association more than its *blessing*.'

Raymund Theron

115 Medfontein
Bloemfontein

1. Correspondence (1961): S. Afr. Med. J., 35, 550.